THE Journal of the Kansas Medical Society

NOVEMBER
1956

VOL LVII
NO XI

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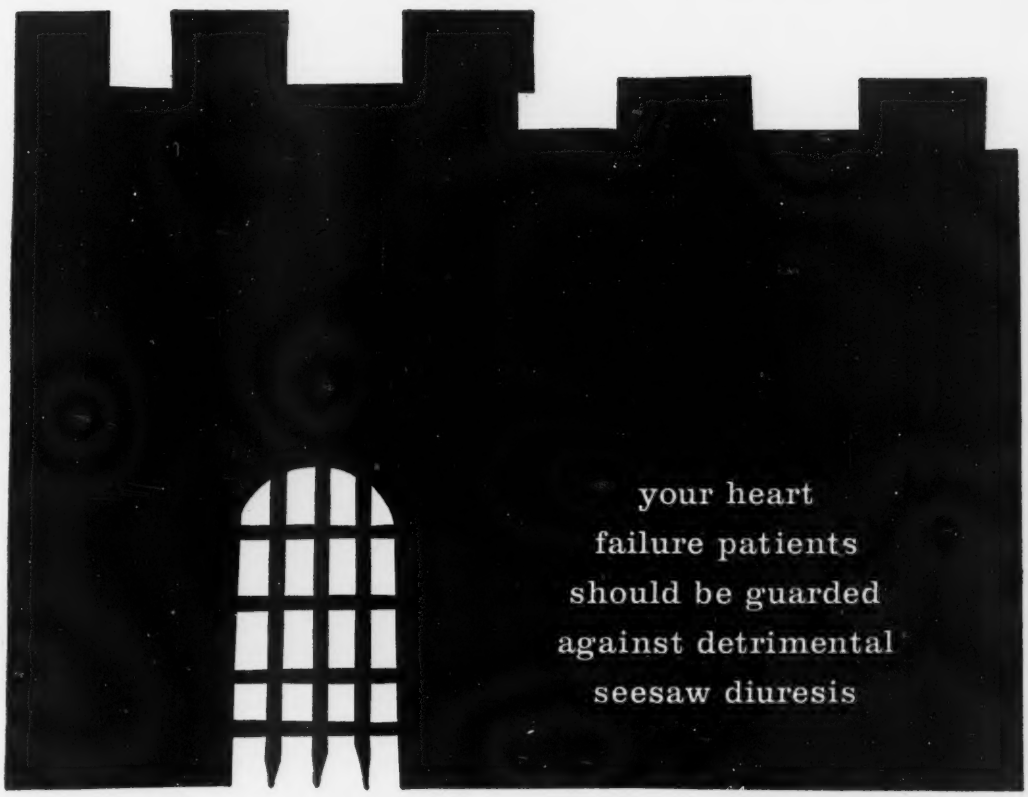

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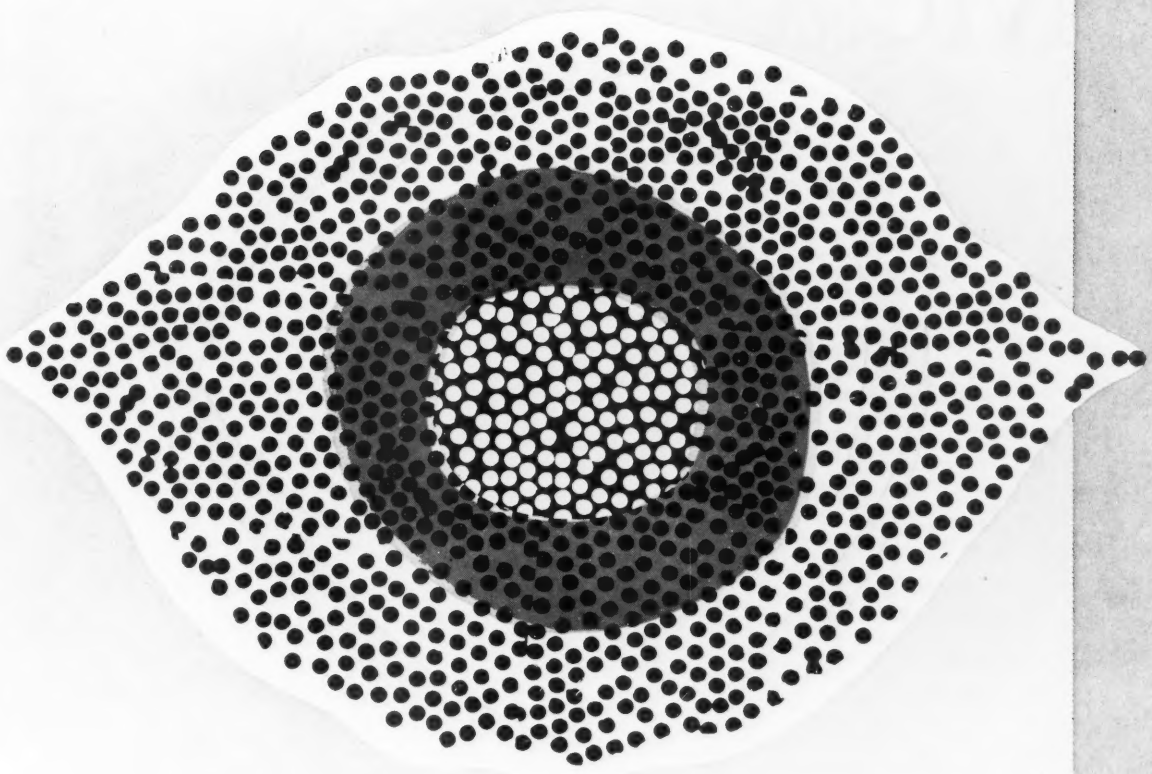

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Good Nutrition and Endocrine Functioning

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1. Ralli, E. P., and Dumm, M. E.: The Hormonal Control of Metabolism, in Wohl, M. G.: Modern Nutrition in Health and Disease, Philadelphia, Lea and Febiger, 1955, pp. 57-74.
2. McHenry, E. W.: Nutrition and Endocrine Function, Borden's Review of Nutrition Research, *16*:17 (Mar.-Apr.) 1955.
3. Ershoff, B. H.: Conditioning Factors in Nutritional Disease, Physiol. Rev. *28*:107 (Jan.) 1948.
4. Keys, A.; Brozek, J.; Henschel, A.; Mickelsen, O., and Taylor, H. L.: The Biology of Human Starvation, Minneapolis, University of Minnesota Press, 1950.
5. Samuels, L. T.: Progress in Clinical Endocrinology, New York, Grune and Stratton, 1950, p. 509.

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

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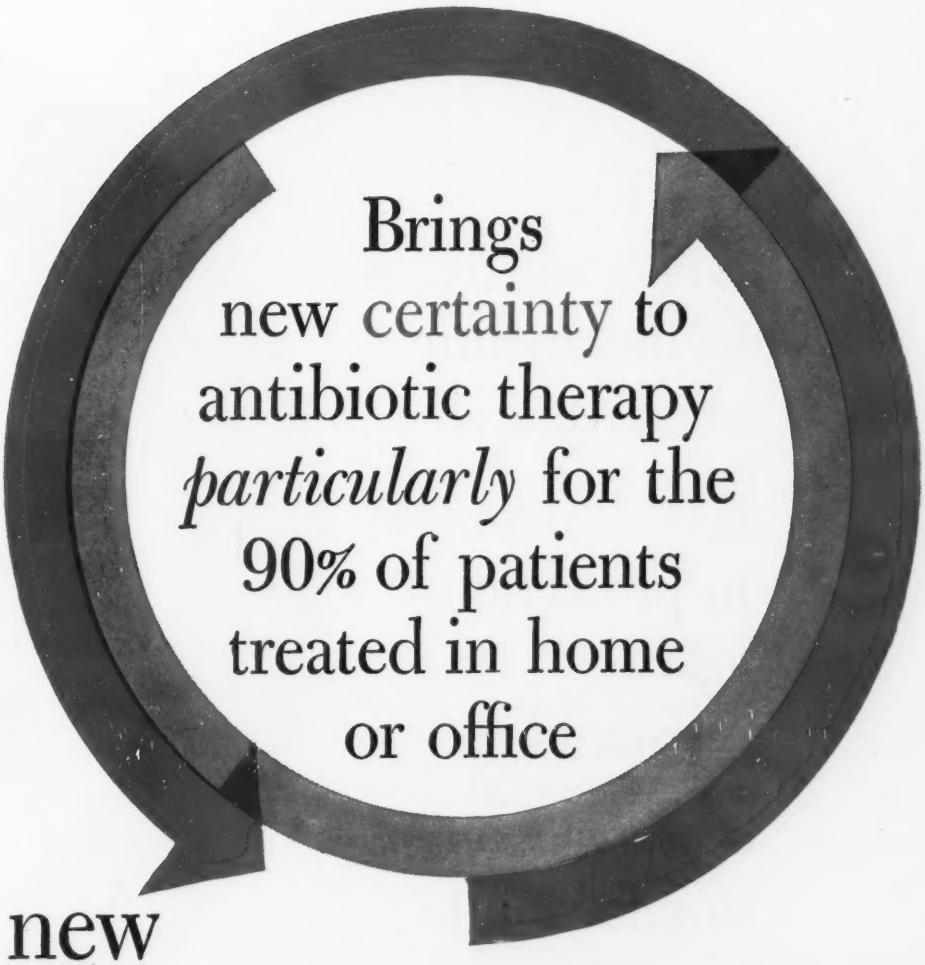

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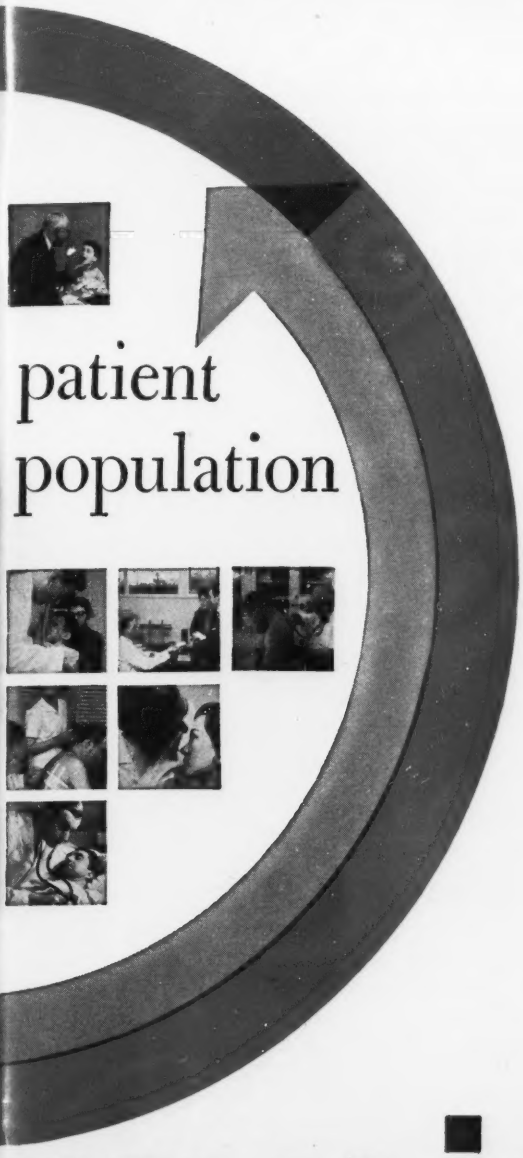

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a new certainty

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Superior control of infectious diseases through superior control of the changing microbial population is now available in a new formulation of tetracycline, outstanding broad-spectrum antibiotic, with oleandomycin, Pfizer-discovered new antimicrobial agent which controls resistant strains. The synergistic combination now brings to antibiotic therapy: (1) a new fuller antimicrobial spectrum which includes even "resistant" staphylococci; (2) new superior protection against emergence of *new* resistant strains; (3) new superior safety and toleration.

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Pfizer

superior control of infectious disease through superior control of the changing microbial population

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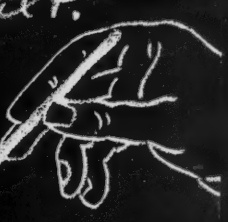

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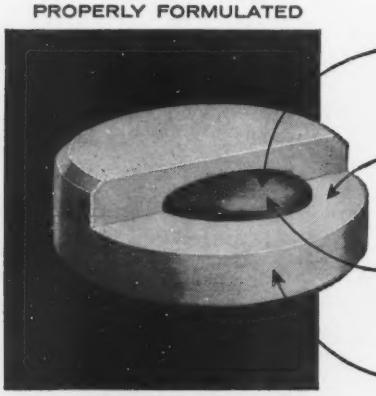

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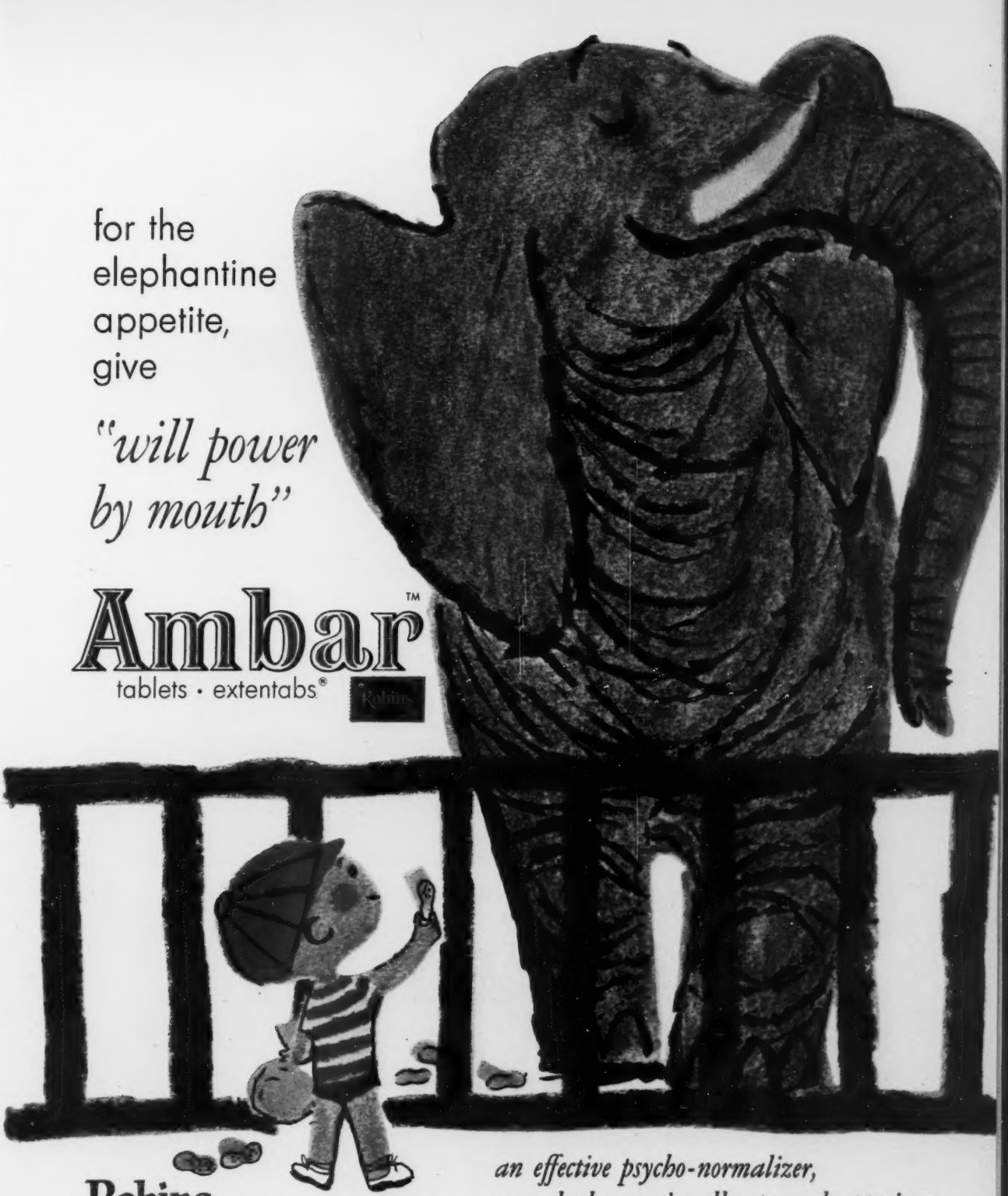

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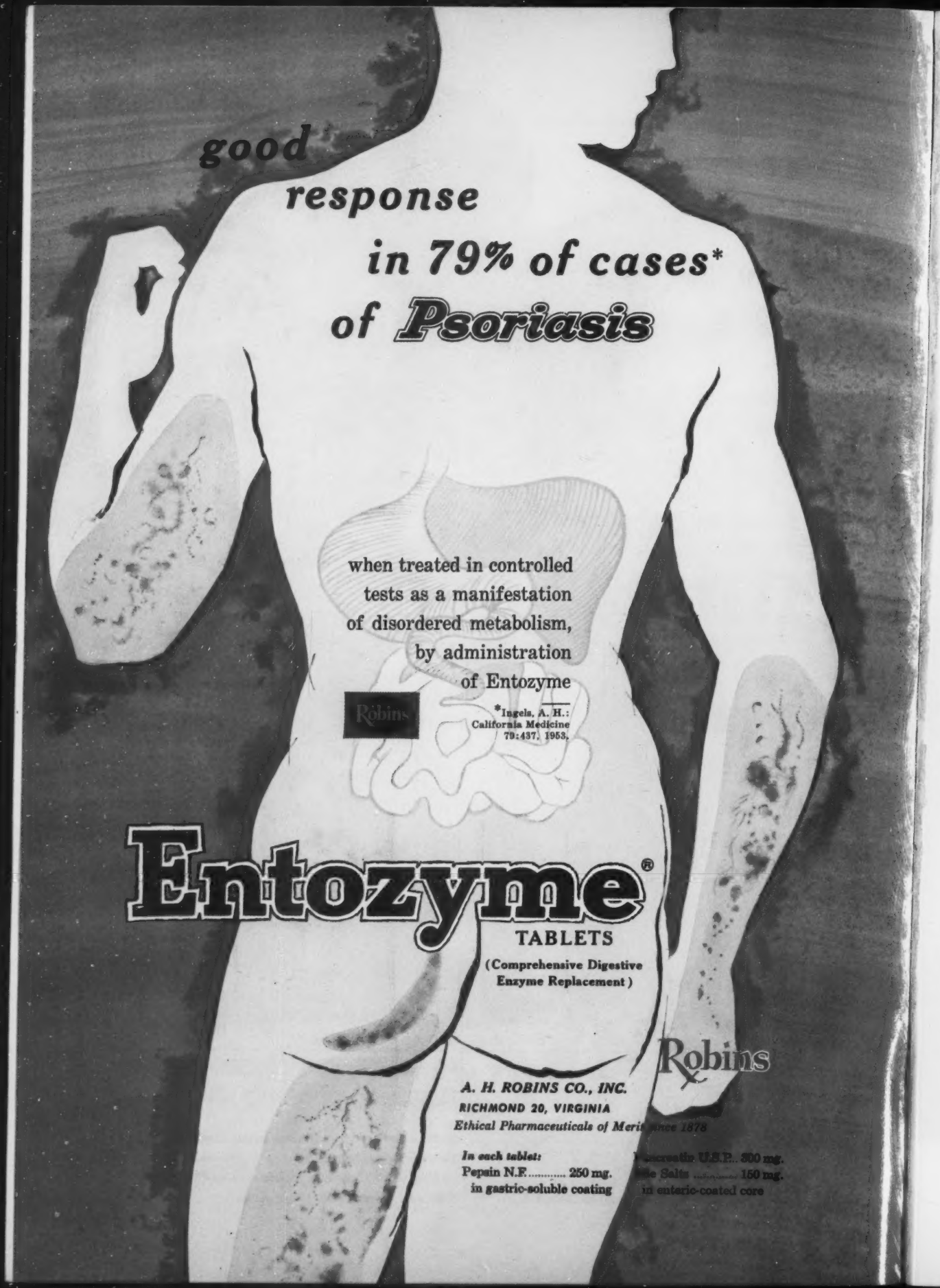
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In one prescription the one antibiotic product most likely to be effective

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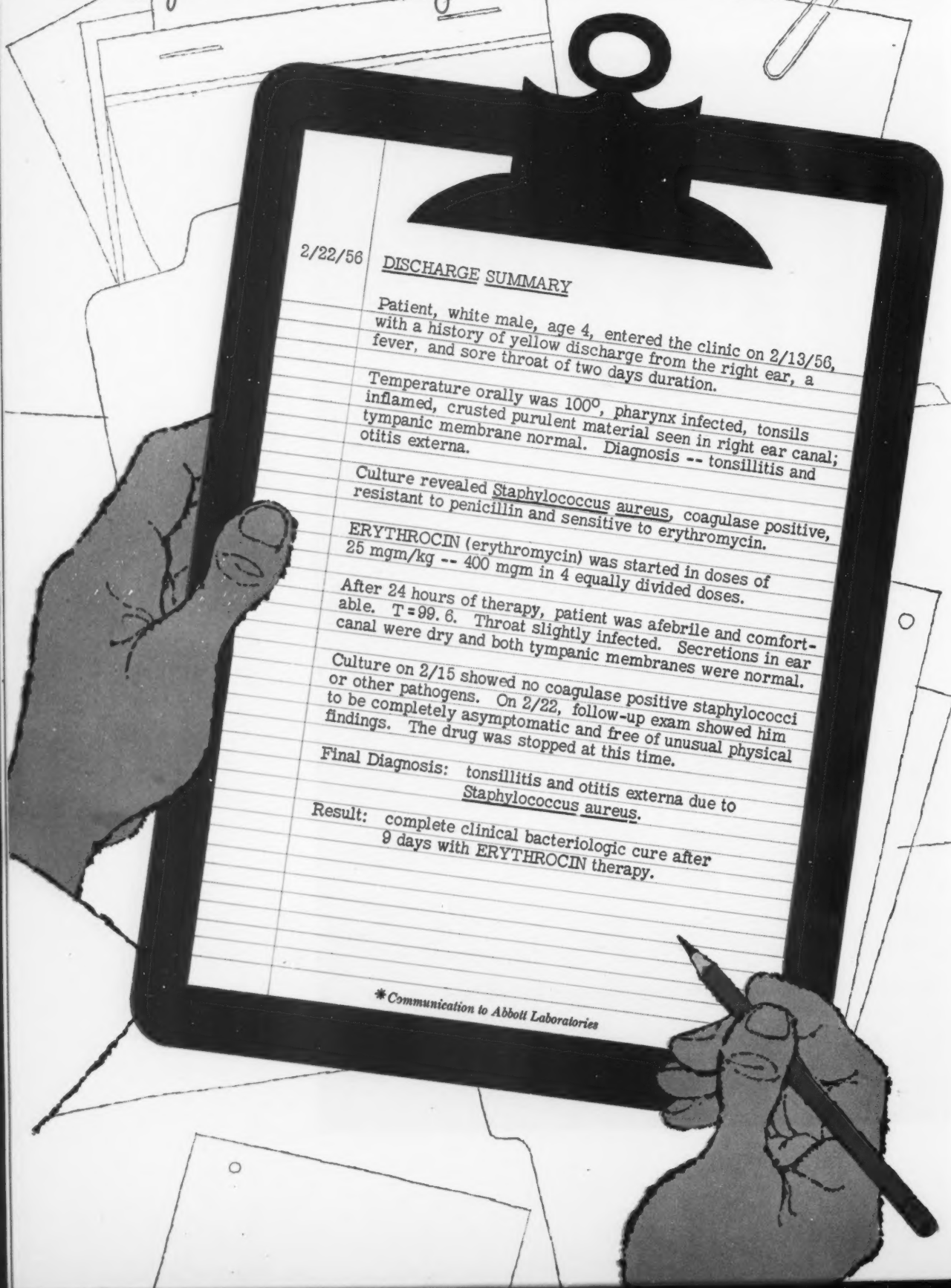
Erythrocin in treating tonsillitis and otitis externa*



2/22/56

DISCHARGE SUMMARY

Patient, white male, age 4, entered the clinic on 2/13/56, with a history of yellow discharge from the right ear, a fever, and sore throat of two days duration.

Temperature orally was 100°, pharynx infected, tonsils inflamed, crusted purulent material seen in right ear canal; tympanic membrane normal. Diagnosis -- tonsillitis and otitis externa.

Culture revealed Staphylococcus aureus, coagulase positive, resistant to penicillin and sensitive to erythromycin.

ERYTHROCIN (erythromycin) was started in doses of 25 mgm/kg -- 400 mgm in 4 equally divided doses.

After 24 hours of therapy, patient was afebrile and comfortable. T = 99.6. Throat slightly infected. Secretions in ear canal were dry and both tympanic membranes were normal.

Culture on 2/15 showed no coagulase positive staphylococci or other pathogens. On 2/22, follow-up exam showed him to be completely asymptomatic and free of unusual physical findings. The drug was stopped at this time.

Final Diagnosis: tonsillitis and otitis externa due to Staphylococcus aureus.

Result: complete clinical bacteriologic cure after 9 days with ERYTHROCIN therapy.

*Communication to Abbott Laboratories

"clinical response good or excellent"

In one recent study, 18 patients with acute follicular tonsillitis and septic sore throat, were given erythromycin. Infecting organism was *Str. pyogenes*. The investigator stated, *"In all 18, the clinical response could be regarded as either good or excellent."*[1]

This, of course, is only one of many reports showing the effectiveness of ERYTHROCIN against coccic infections. You'll get the same good results (nearly 100% in common, bacterial respiratory infections) when your prescription reads *Filmtab* ERYTHROCIN Stearate.

"toxicity lower in erythromycin-treated patients"

After a study of 208 patients treated with erythromycin (78), procaine penicillin (78) and a placebo (52), the investigator stated: *". . . the incidence of toxicity* (compared to procaine penicillin) *was significantly lower in the erythromycin-treated patients."*[1]

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1. Herrell, W. E., Erythromycin, Antibiotics Monographs, No. 1, p. 29, New York, Medical Encyclopedia, Inc., 1955.
Idem p. 30.

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ATARAXOID is a unique, new combination of STERANE and ATARAX, which now permits simultaneous symptomatic control and reduction of attendant anxiety and apprehension in rheumatoid arthritis and other indications.

The added tranquilizer control, desirably easing mental stress, also directly assists clinical progress. It minimizes the chance of exacerbation related to emotional strain and facilitates patient confidence and cooperation in the therapeutic program toward maximum rehabilitation.

ATARAXOID exerts the anti-rheumatic, anti-inflammatory activity of STERANE distinctly superior to previous steroids, effective in radically reduced dosage, and with minimal disturbance of electrolyte and fluid metabolism.

The ataractic effect is a central neuro-relaxing action – the result of a marked cerebral specificity – free of mental fogging and devoid of any major complications: no liver, blood or brain damage. This peace-of-mind component is also used in the lowest dosage range.

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prednisolone and hydroxyzine

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"In 96 per cent (24 patients) clinical symptoms were relieved and the blood pressure maintained at comfortable levels. . . ."[1]

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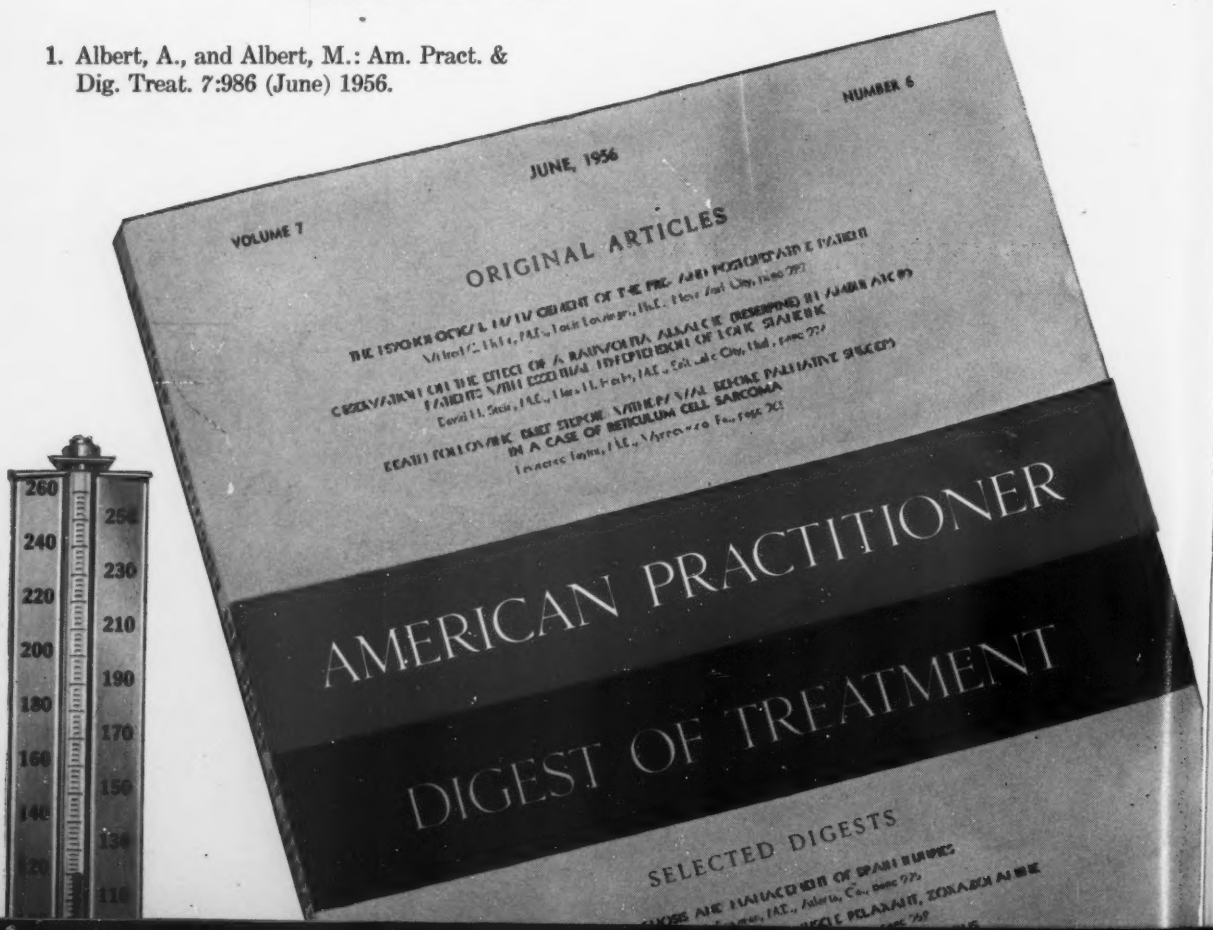
Philadelphia 1, Pa.

1. Albert, A., and Albert, M.: Am. Pract. & Dig. Treat. 7:986 (June) 1956.



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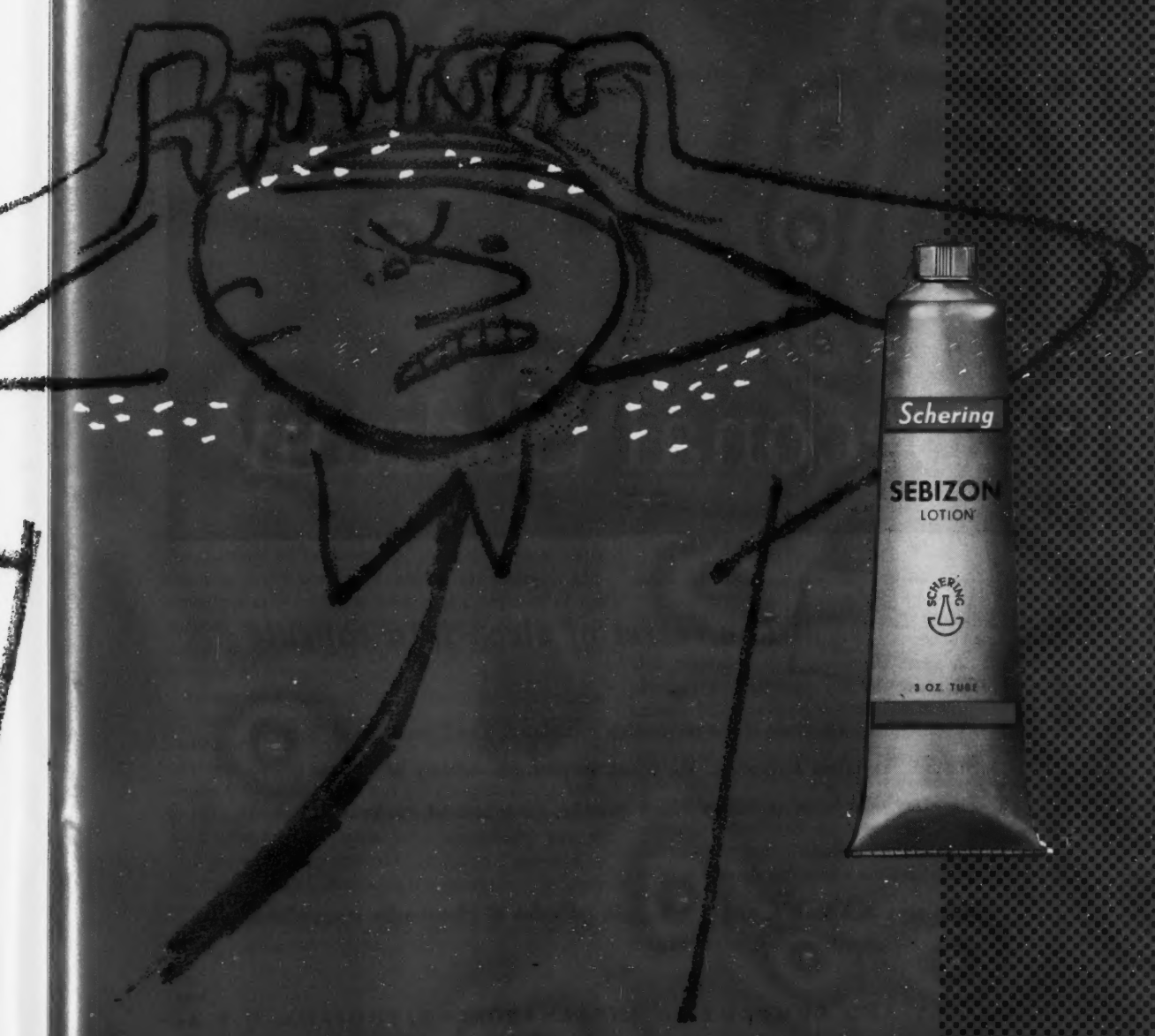

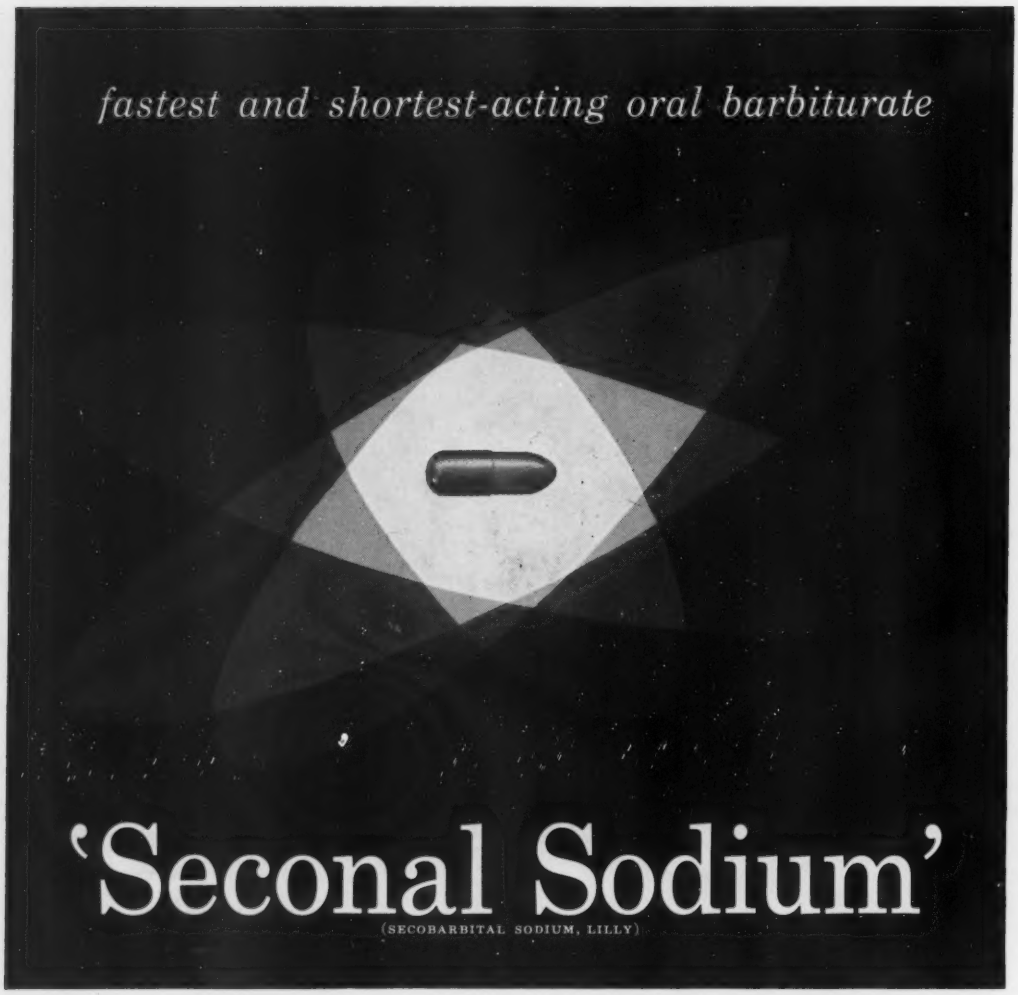

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Colon

The Selection of Operation for Carcinoma of the Colon

PETER A. ROSI, M.D., *and* NICHOLAS J. CAPOS, M.D., *Chicago*

Because of its segmental blood supply and lymph drainage, the colon lends itself to radical resection for carcinoma. Physiologically, long segments of colon can be removed without any apparent disturbance of bowel function. In view of these facts, in planning operations for carcinoma of the colon, every attempt should be made to remove the tumor and its region of local spread and lymph node metastases. In order to obtain the greatest number of surgical cures for carcinoma of the colon, wide resection of the tumor and its avenues of spread should be done in all cases.

Radical resection of malignant tumor is accepted in principle by all surgeons. However, there is no uniform opinion as to the extent to which an operative procedure is to be carried in order to effect a cure. Resectability of carcinoma of the colon, particularly the large, often fixed, and infiltrating tumors, is governed not only by the training and experience of the surgeon but also by the acceptance of the fact that today, at least, surgery is the only hope for a cure or relief of symptoms.

In our own series of 385 patients with carcinoma of the colon operated upon in the period between 1940 and 1950, the carcinoma was resectable in 330, or a resectability rate of 86.6 per cent. The mortality rate in the resected cases was 3.3 per cent. In the 1950 to 1953 period there were 149 patients, of whom 144 were treated by resection, or a resectability rate of 96.6 per cent and a mortality rate of 5.5 per cent in the resected cases. Although the mortality is 2.2 per cent greater in the 1950 to 1953 period, during which time the tumors were resected radically, our resectability rate increased 10 per cent, thereby giving an additional 7 to 8 per cent of operated patients greater comfort, if not a greater possibility of cure.

Presented at the 97th annual session, Kansas Medical Society, Topeka, May 2, 1956.

PREOPERATIVE PREPARATION AND ANESTHESIA

The generally accepted plan of preparing patients for colon surgery is by improving their general condition through high protein, high carbohydrate, low residue diet and multiple blood transfusions whenever necessary. The preoperative use of antibiotics, either the relatively nonabsorbable sulfonamides, namely Sulfasuxidine and Sulfathalidine, or one of the broad-spectrum antibiotics such as Aureomycin, terramycin, neomycin or Chloromycetin, in order to reduce the number of organisms in the colon, has added greatly to the safety of colon surgery. These drugs reduce the hazards of infection and have been responsible for the great reduction of morbidity and mortality. Although antibiotics are available today which will reduce the number of bacteria in the colon to low and clinically insignificant concentrations, evidence of toxicity has been demonstrated.

Factors determining choice of operation are discussed, along with pre-operative preparation, anesthesia, and surgical procedures effective in various areas of the colon.

In our series there seemed to be little if any difference in postoperative morbidity and mortality between the preoperative use of Sulfasuxidine and Sulfathalidine, the preoperative administration of neomycin, Aureomycin, terramycin and Chloromycetin, alone or in conjunction with Sulfasuxidine. In the group of patients who were given either Aureomycin, terramycin or neomycin, we had three cases of pseudomembranous colitis, one of which proved fatal, and one fatal case of diffuse jejunitis, ileitis and colitis.

While these conditions had been known to occur before the introduction of antibiotics, the increased incidence of these complications seems to be more than a coincidence. The broad-spectrum antibiotics are invaluable and often life-saving; however, their use should be restricted to definitely established infections. They should not be used indiscriminately. Until less toxic drugs are available, it seems that the relatively nonabsorbable drugs such as Sulfasuxidine or Sulfathalidine, which are associated with fewer toxic sequelae, are more suitable for the preparation of the colon for surgery.

In the preparation of the gastrointestinal tract for surgery there is great need for an agent which will destroy the desquamated cancer cell in much the same way the antibiotics suppress bacterial activity. Tumors in the gastrointestinal tract give off isolated or clumps of cancer cells which lie free in the lumen of the bowel. These cells are frequently viable and can be locally transplanted during the surgical procedure, particularly if the bowel is resected and an open anastomosis is done. Much can be done to prevent local seeding of malignant cells during operation by careful attention to asepsis and discarding all instruments, needles, gloves, and drapes potentially contaminated by cancer cells; or by mechanically removing desquamated cells by rectal lavage just prior to a colon-rectal anastomosis. However, some viable malignant cells undoubtedly are spread locally and under suitable conditions grow into residual tumors.

Anesthesia has played an important role in the reduction of mortality and morbidity following colon surgery. There seems to be little difference in morbidity and mortality in the use of general as contrasted with spinal anesthesia. However, too little attention has been given to local anesthesia in the so-called poor risks, elderly and debilitated patients. It is surprising how frequently an extensive intestinal resection can be done under local procaine anesthesia and with little shock to the patient.

FACTORS DETERMINING CHOICE OF OPERATION

The choice of operation for carcinoma of the colon is determined by the site of the tumor, its local spread, and regional lymph node drainage. Clinicopathological studies of Westhues, Gilchrist and David, Duke and Waugh have established the course of lymph node spread of carcinoma of the colon and also determined the rather limited spread of the carcinoma in the bowel wall, either proximally or distally to the tumor. As a result of these studies there has developed a generally uniform opinion among surgeons as to the amount of bowel which should be removed in order to eradicate the local spread of the malignancy in the bowel wall itself. There is no such uniformity of opinion as to the extent of lymph node resection necessary in order to effect the greatest number of cures. It may be that the disparity in statistics as to five or more years cure of cancer of the colon is a result of differences in the extent of resection of the potentially involved regional lymph nodes. These differences may occur between various segments of colon and between the results obtained by different surgeons.

In our follow-up studies of patients operated upon for cancer of the colon, the five-year survivals were better on the right than on the left side of the colon. Since the type of tumor and method of spread are essentially alike in all segments of the colon, it was felt that the extent of resection of the potentially involved lymph nodes was probably the cause of the fewer cures on the left as opposed to the right colon. In order to improve our postoperative curability of cancer of the colon, particularly on the left side, we have extended resections of the colon and mesentery so as to include the greatest number of lymph nodes that drain the involved segment of bowel.

CHOICE OF OPERATION IN VARIOUS AREAS

Cecum. Tumors of the cecum spread predominantly along the ileocolic group of lymph nodes. The greatest number of cures in this group of tumors can be obtained by a right hemicolectomy with resection of the terminal 20 cm. of ileum and the right colon along with their entire lymph node containing mesentery up to the midcolic artery. The continuity of the bowel is re-established with an end-to-end, end-to-side, or side-to-side anastomosis, whichever is most suitable.

Ascending Colon and Region of Hepatic Flexure. Tumors of the ascending colon and region of the hepatic flexure spread predominantly along the right colic and middle colic group of nodes. Resection of the right colon and transverse colon with all the lymph nodes along the right and middle colic arteries is a necessity in order to remove tumor and resectable lymph node metastases in this area. The continuity of the bowel is re-established with an anastomosis between the ileum and colon in the region of the splenic flexure.

Midtransverse Colon to Region of Splenic Flexure.

Tumors from the midtransverse colon to the region of the splenic flexure metastasize to lymph nodes that lie along an arterial arcade that reaches from the middle colic to the left colic arteries. From these chains of nodes, the tumor cells may reach the superior mesenteric, inferior mesenteric, and periaortic group of lymph nodes. In order to remove the tumor and the metastatic carcinomatous lymph nodes, the operation of choice would be a resection of the ascending, transverse, and descending colon with their entire group of regional nodes. The ileocolic, right, and middle colic arteries are ligated at their origin from the superior mesenteric artery; the left colic artery is ligated at its origin from the inferior mesenteric artery, and the lymph nodes around the inferior mesenteric artery and aorta are excised. The continuity of the bowel is re-established by anastomosing the ileum to the lower sigmoid colon.

Left Colon. Tumors of the left colon, from descending colon to rectum, have a definite pattern of upward spread from the regional lymph nodes to the lymph nodes around the inferior mesenteric artery and aorta. While the number of patients who have carcinomatous lymph node metastases to inferior mesenteric and periaortic groups of nodes and who have curable cancer has not been established and can be determined only by prolonged follow-up studies, operation for left colon carcinomas should include, with resection of the tumor, this entire lymph node bearing area.

In a study of 36 patients with carcinoma of the left colon in which lymph node spread was determined in formalin-fixed, surgically resected specimens, three patients, or eight per cent, had involved nodes along the inferior mesenteric artery from its origin at the aorta to its bifurcation, or approximately within 1 cm. of the aorta. Five patients, or 14 per cent, had carcinomatous nodes within 2 cm. of the aorta, and 8 patients, or 22 per cent, had malignant cells in nodes 3 cm. from the aorta. The increased amount of surgical trauma that results from the dissection of periaortic nodes and ligation of the inferior mesenteric artery does not affect postoperative morality or morbidity.

In our own series there were 119 patients with limited segmental resections of colon with one death and 49 patients with division of the inferior mesenteric artery at the aorta and dissection of periaortic nodes which reservedly is called left hemicolectomy, with no deaths. There were 136 patients who had a Miles type of abdominoperineal resection with 6 deaths, or a mortality rate of 4.4 per cent, and 55 patients who had an abdominoperineal resection with left hemicolectomy with three deaths, or a mortality rate of 5.5 per cent. The differences in morbidity and mortality rates between the limited segmental resections of the left colon and the extended resection or left hemicolectomy are statistically insignificant, so as to warrant the performance of a left hemicolectomy in all patients with left colon carcinoma.

Descending Colon and Sigmoid. Tumors of the descending colon and sigmoid metastasize to the regional lymph nodes, to the inferior mesenteric group of nodes, and to the nodes along the aorta. The operation of choice for this group of carcinomas is a resection of the left colon with its entire lymph node containing mesentery from the splenic flexure to the rectum, that is, left hemicolectomy. The continuity of the bowel is re-established by an anastomosis of the colon from the region of the splenic flexure to the rectum.

Rectosigmoid. Tumors in the region of the rectosigmoid, as shown by histological studies of resected tumor specimens, spread predominantly upward along the inferior mesenteric group of lymph nodes with little tendency to spread retrograde to lymph nodes below the lower edge of the tumor. Carcinomatous involved nodes are rarely found more than 4 cm. below the lower border of the tumor, although isolated instances are recorded in which metastatic carcinoma was found in lymph nodes that lay 10 to 15 cm. below the tumor. The results of these histological studies of resected specimens have been accepted by surgeons as guides in determining the length of colon to be resected proximal and distal to the carcinoma. Very little attention has been drawn to the fact that these studies were done on formalin-fixed specimens which may have contracted considerably as a result of the action of formalin.

In the transference to distances obtained from the study of formalin-fixed tumors back to the living patient, allowances must be made for the amount of contraction of the bowel after it is resected, which often is as much as 50 per cent, and allowance made for further retraction, if the bowel is fixed in formalin without first being pinned to a board so to maintain its original size. Since the length of resected bowel is rarely measured in its living state in the patient and in its fixed state in order to obtain the factor of contraction, the distances obtained in histological studies of resected specimens of bowel carcinomas are only approximate; they may often be only half to one-third the distances in the respective patient. If histological distances are interpreted without the knowledge of amount of specimen contraction, then they cannot be safely applied to the patient without first empirically assuming a contraction factor of two to three. If, for example, a carcinomatous lymph node is found 4 cm. below the tumor in an unpinned formalin-fixed specimen, in the patient this distance would probably have been about 8 to 12 cm. below the tumor.

The conflicting results of the high incidence of

local recurrences following segmental resection of carcinomas of the rectosigmoid in the face of limited retrograde spread in histological studies or resected specimens is probably due in part to the direct application of uncorrected histological distances to the patient with the result that local, probably resectable carcinomatous tissue is left in the patient. Also, underestimating the extent of retrograde spread of carcinoma may cause carcinomatous lymph nodes to be divided and malignant cells to be seeded locally. Because of these two potential dangers, it has been our practice never to attempt to do a segmental resection of carcinoma of the rectosigmoid unless 12 to 15 cm. of bowel with its mesentery, below the tumor, can be removed.

Tumors of the rectosigmoid spread through the lymph nodes along the superior hemorrhoidal, sigmoid, and inferior mesenteric arteries with possible retrograde spread to the lymph nodes as low as 10 to 12 cm. below the tumor. The entire left colon, up to the region of the splenic flexure, the regional lymph nodes up to and including the periaortic nodes around the origin of the inferior mesenteric artery, and about 15 cm. of bowel with its mesentery below the tumor should be resected in order to obtain the greatest number of cures.

The continuity of the bowel is re-established by an anastomosis between the colon from the region of the splenic flexure to the lower rectum. In those patients in whom it is not possible to remove from 12 to 15 cm. of bowel below the lower edge of the tumor, an abdominoperineal resection with left hemicolectomy is done.

Rectum. Carcinoma of the rectum, because of its location in the midst of the rich lymphatic network in the pelvis, spreads upward along the superior hemorrhoidal to the inferior mesenteric and periaortic nodes, but it also spreads laterally along the middle hemorrhoidal, hypogastric, and iliac group of lymph nodes. Because of this wide local dissemination of carcinoma of the rectum, complete eradication of the tumor tissue is difficult to accomplish.

Of the patients who presented themselves with residual carcinoma following abdominoperineal resection, as shown by Diddish, in 50 per cent the carcinoma was limited to the structures of the pelvis at the initial operation. The residual carcinoma was found especially in the posterior vaginal wall and in the fascia posterior to the prostate and seminal vesicles. Because of the extensive local spread of carcinoma of the rectum, operation should be planned so as to remove all the regions of tumor infiltration and metastases.

The operation of choice for rectal carcinomas is an abdominoperineal resection with resection of the mesentery of the left colon up to the origin of the inferior mesenteric artery, which is divided from the aorta, and dissection of the periaortic lymph nodes. The colon in the region of the splenic flexure is brought out as a colostomy through a stab wound in the left upper quadrant.

Since metastatic carcinoma is found in lymph nodes along the middle hemorrhoidal, obturator, hypogastric, and iliac chain of lymph nodes in as high as 24 per cent of cases of advanced carcinomas of the rectum, abdominopelvic node dissection should be performed more frequently than it has been heretofore with the hope of increasing the curability of this group of tumors.

Carcinoma of the rectum which has extended into the pelvic urogenital system presents particular problems. In females in whom there is infiltration of the posterior vaginal wall or uterus by rectal carcinoma, resection of the genital tract, that is, ovaries, tubes, uterus, and vagina, along with the attached tumor, is necessary in order to give the greatest chance for a cure.

In males, extension of the rectal cancer into the urinary bladder may require a local resection of the bladder. Infiltration of the seminal vesicles and prostate may require excision of these organs, if the extension seems limited to them. If, however, tumor tissue has extended beyond these structures, complete removal of the pelvic viscera with implantation of the ureters into the bowel or on the skin, gives the patient his only hope for the relief of his symptoms or possible cure.

Patients who have undergone pelvic exenteration for rectal cancer have little difficulty in adjusting to the new way of life. The relief from pain and rectal bleeding more than compensates for the inconvenience of a wet colostomy or multiple abdominal wall stomata that result from transfer of the ureters to the colon, ileal conduit, artificial bladder, or to the skin.

Pelvic exenteration is a recent development in the treatment of advanced rectal cancer, and there is still insufficient statistical data to determine its role in the treatment of these infiltrating tumors. With new developments in surgical techniques and postoperative care, the operative mortality for pelvic exenteration is about 10 per cent, which is an acceptable mortality rate for a fatal disease. Pelvic exenteration, whenever possible to perform, is a more acceptable operation than the so-called palliative colostomy with the tumor left in situ. Palliative colostomy, except for the relief of symptoms of bowel obstruction, offers little or no relief from the ravages of this infiltrating tumor.

30 North Michigan Avenue
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Plaster Casts

Application of Splinting Agents for Immobilization of Injuries

GEORGE E. OMER, JR., M.D., ***Fort Riley***

From the time of ancient Egyptian history, many different substances have been recorded as splinting agents for immobilization of injuries. As late as the last half of the 19th century, a list of agents used for stiffening mesh bandage fracture dressings would include starch, paraffin, glue, gum thickened with powdered chalk, silicate of soda, white of egg stirred with flour to form stiff batter, and plaster of Paris. Today the plaster of Paris bandage cast is the most versatile material in common use for immobilization of a fracture.

Plaster of Paris was used by the Arabians long before it was introduced as a casting agent in Europe early in the 19th century. The early casting technique consisted of placing the injured limb in a trough into which the liquid plaster was poured. After the plaster dried, the limb and its encasing moulded cast was broken free from the trough. This awkward method was replaced in 1852 when Antonius Mathijsen, a Flemish Army surgeon, devised the method of using a mesh bandage impregnated with plaster of Paris. Two years later the plaster bandage was used extensively in the Crimean War and became the standard technique for fracture immobilization.

Plaster of Paris is produced by heating gypsum, or calcium sulfate, until the water of crystallization evaporates. In its natural state, gypsum contains approximately 21 per cent water of crystallization. In the manufacturing process, all but approximately six per cent of the water is evaporated. The dehydrated gypsum is plaster of Paris. When water is added to the dry plaster of Paris powder, the action is reversed and moisture is taken up to form crystals of gypsum. This plaster "setting" mechanism produces considerable heat and is accomplished when the chemical formation and interlocking of the gypsum crystals is completed. Practically, setting is complete when heat is no longer given off from the cast.

Plaster of Paris bandages are manufactured by several reliable companies and practically all plaster of Paris now on the market is consistently good. Luck[4] demonstrated that more unsatisfactory plaster casts are the result of poor technique of application than the result of defective plaster. In an emergency, plaster bandages can be made from ordinary crinoline bandages and dental plaster of Paris. The plaster powder is rubbed into the meshes of the crinoline, and the bandage is loosely rolled. The setting of this substitute plaster bandage can be speeded by adding three or four teaspoonfuls of salt per quart of water. Commercial fast-setting plaster bandages placed in salted water may produce too much heat and result in a burned patient.

Many failures in the treatment of fractures can be traced to inadequate application of plaster casts. Good fracture results involve careful attention to position, padding, equal distribution, and uniform thickness of plaster bandages.

A plaster cast should be applied while the patient is lying down on a narrow slightly padded table that is a convenient height for the surgeon. If the patient watches the rolling of the plaster bandages, it becomes difficult for him to relax. Plaster cast application is thus best performed with the relaxed patient lying down and his interest diverted from the operation as much as possible.

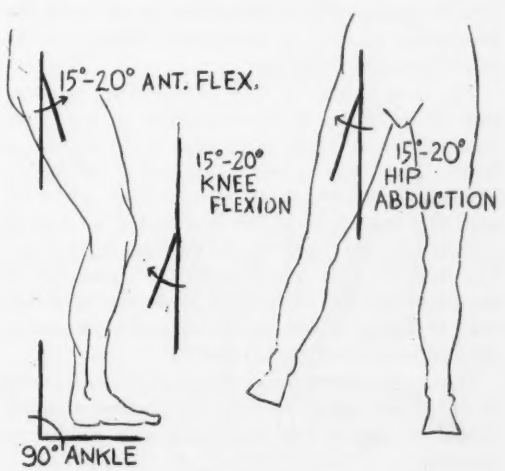

Figure 1. The functional position of the lower extremity.

The author, formerly of Kansas City and now a major in the Army Medical Corps, is serving as chief of Orthopaedic Surgery Service at U. S. Army Hospital, Fort Riley.

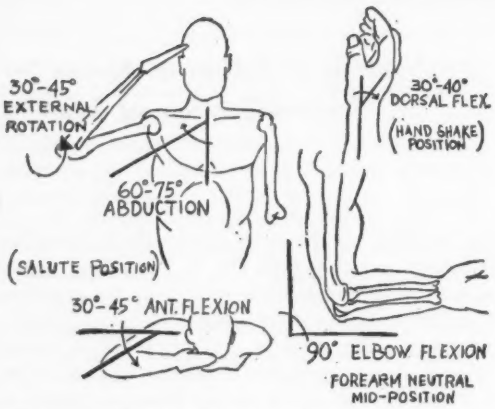

Figure 2. The functional position of the upper extremity.

In applying a plaster cast the surgeon holds the extremity or occasionally supports it by a special apparatus such as an overhead sling. It is better for the more experienced surgeon to support and position the injured extremity while the less experienced operator applies the plaster rolls. If the lower extremity is involved, two assistants are required for the important functional angles of the hip, knee, and tarsal joints. The most important single feature in the proper application of a plaster cast is to secure the proper functional position of the injured part.

The functional position of the ankle joint is 90 degrees or at right angles with the long axis of the leg. The other tarsal joints should be moulded to maintain a normal arch and to prevent rotation of the foot from its normal axis. The knee joint should be placed at 15 degrees of flexion from a fully extended position. If the hip joint is included in a spica-cast, the joint should be in 15 to 20 degrees of flexion and 15 to 20 degrees of abduction.

The wrist should be in a hand-shaking position, with 30 to 40 degrees of dorsiflexion at the carpal joint and the interphalangeal joints in 45 degrees of flexion. The elbow is best maintained at 90 degrees of flexion or at right angles with the long axis of the arm. The forearm is placed in a neutral or midline position with the thumb up and the little finger down. The shoulder joint should usually be maintained in the salute position, with 60 to 75 degrees of abduction, 45 degrees of anterior flexion, and 45 degrees of external rotation (Figures 1 and 2).

During the entire period of application and setting of the plaster cast, all joints of the involved extremity should be aligned and maintained in a position of function.

A protective padded covering for the skin is applied after the desired position and alignment of the involved extremity have been secured. The protective padding and the overlying plaster bandage should extend proximal or above and distal or below the first major joint adjacent to the injury (Figure 3). Thus, a tibial fracture should be immobilized in a long leg cast that includes the foot and the thigh.

If the injury is acute with associated hematoma and edema of the extremity, a layer of sheet wadding should lie next to the skin as padding. Sheet wadding is a thin non-absorbent cotton batting. It is applied by rolling it on the limb, rather than pulling or twisting, and it should accurately conform to the contour of the extremity. Bony protuberances and subcutaneous nerves should receive extra padding (Figure 4). The added padding should be carefully placed. Displaced or bulky padding is not only ineffective but may result in increased chafing and skin irritation.

If swelling is not anticipated, stockinette may be applied as the immediate skin covering. Stockinette is a seamless tubular knit cotton fabric. The proper

Figure 3. A long leg cast. Note the rounded-arc walker and toe plate for protection during ambulation.

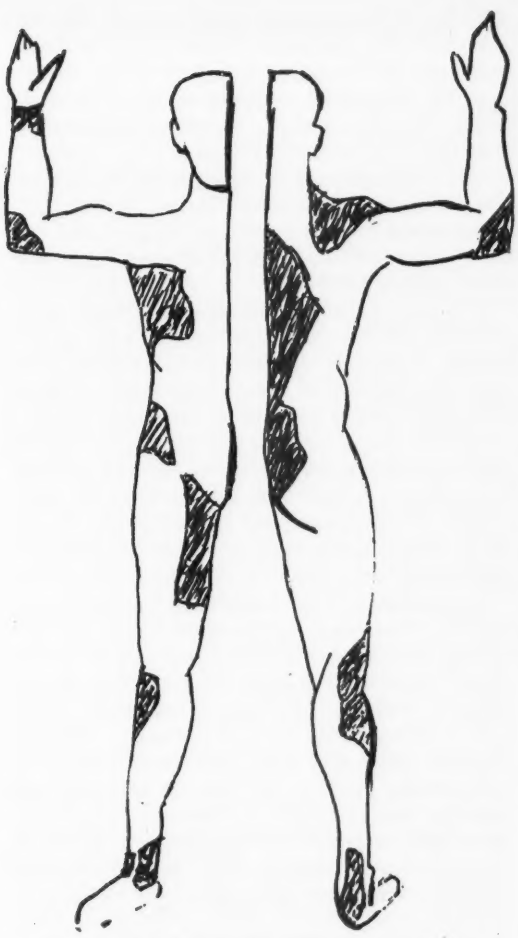

Figure 4. The body areas that require protective padding.

width stockinette is pulled over the limb with special care to smooth all wrinkles. Spot padding over stockinette is usually composed of soft wool felt or foam rubber of medium density. All spot padding should be anchored with sheet wadding before the actual wrapping of the plaster bandage is accomplished.

After the extremity has received protective padding, an appropriate width of plaster bandage should be selected. For example, a plaster bandage six inches wide is too bulky for use on any extremity of a six weeks old child.

The selected bandage roll is placed on one end in water deep enough to cover the entire bandage. Sufficient water should be available so that it may be changed after every few rolls of plaster and kept clear and clean. Cool water retards setting time and warm water accelerates the setting time, so the temperature of the water should depend on the operator and his technical speed of cast application. The bandage roll is saturated with water when bubbles no longer rise to the surface. The roll is then grasped at both ends and lifted from the water. Excess water is gently squeezed from the roll by pushing both hands toward the middle. The bandage roll should not be bent or twisted (Figure 5). The roll should be squeezed out over a separate container since excessive plaster particles collected in the bottom of the water basin will act as "feeding agents" and accelerate the setting time of the plaster.

The loose leading edge of the plaster roll is fitted to the extremity and then rolled onto the limb like a rug. The roll of plaster bandage remains in contact with the surface of the limb and is not lifted, twisted, or placed under tension or pull. The bandage should be allowed to turn in the direction in which it will lie flat, and the plaster is advanced down the extremity by overlapping each turn of the bandage by approximately half its width. Each turn is applied carefully, and the surgeon's hands should follow the natural inclination of the bandage roll without forcing it into a twisted position. At tapering parts of the limb the turns are made to lie evenly by utilizing small "tucks" that are made in the redundant edge of the

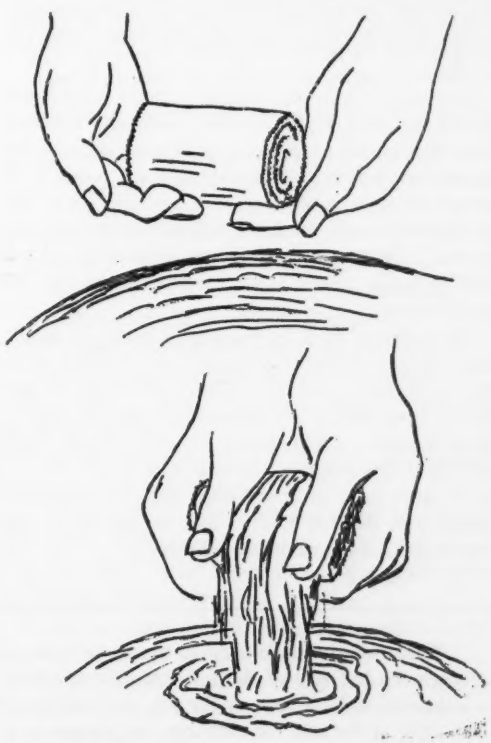

Figure 5. Technique of handling moist plaster rolls.

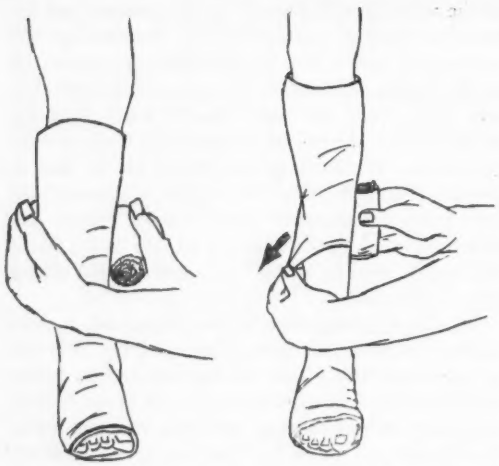

Figure 6. Technique of rolling plaster bandages.

plaster roll with a quick movement of the index finger before each turn is moved into final position (Figure 6). While the major hand is engaged in the rolling-on process, the flat of the minor hand is constantly smoothing and rubbing the plaster. The durability of the cast and its strength for any given weight depend on the welding together of the individual plaster turns by these smoothing movements of the hands.

The surgeon and his assistants should support the plaster cast with the flat of their hands and not with their fingers. Finger pressure produces ridges in the plaster and results in uneven cutaneous pressure. It cannot be over-emphasized that internal folds and wrinkles in a cast result in pressure sores and tissue necrosis. The hands should move constantly and rub and work the plaster for a smooth consistency and even tension.

The pressure made by the plaster bandages should be evenly distributed since tension on a circular bandage is cumulative and may build up to a point of dangerous constriction. To insure even pressure and to guard against a sharp cutting edge, the plaster bandage should be centrally placed in a deep flexure such as the knee joint. It is also more desirable to cut the plaster roll than to reverse its direction in a deep flexure such as the elbow or knee joints.

No single factor has more influence on the ultimate effectiveness and strength of the plaster cast than the maintenance of immobility during the period of setting. Any attempt at manipulation or realignment during this period results in a greatly weakened cast. Tests conducted by Luck[4] showed a 77 per cent reduction in the strength of plaster casts which were bent during this critical period. During the period of setting a heat cradle is usually not indicated, but the cast should be left uncovered in order that it may dry out as much as possible.

If marked swelling or edema of the extremity is anticipated, the cast should be split in a convenient location, such as the anterior aspect. A second technique is a bivalved or bisected cast where one-half of the cast is easily lifted up for inspection. A split cast can be removed without special tools. If a person with an acute fracture is to be transported, the cast should always be split. It is imperative that all components of the dressing are split; a single turn of sheet wadding against the skin is as constrictive as the entire overlying plaster.

Many failures in the treatment of fractures can be traced to inadequate application of the plaster cast. A good plaster cast can be applied only if the involved extremity is in good functional position, the padded covering of the limb is adequately placed without being bulky, the plaster bandage has been smoothly rolled with equal distribution of pressure and uniform thickness, and the cast has been kept immobile until the plaster has thoroughly "set." Adequate skill in plaster cast application is not to be found in the textbook; it can be learned only by a process of continuous repetition.

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When all the mountains in the world have been scaled, when the poles hold no more secrets, when the last acre of the last continent has been traversed, when, in short, everything on our planet is known and catalogued, the way will still be open for discovery. The world will never be conquered so long as the zest for conquest, for adventure, is in men's hearts. —*Maurice Herzog*

Chlorpromazine and Reserpine

Limitations of Their Use in Office Practice

JOHN A. GRIMSHAW, M.D., ***Topeka***

So much has been said about the value of chlorpromazine and reserpine in the last two years that I feel some discussion of their failures and limitations is appropriate. Conservative estimates indicate that approximately 5 tons of chlorpromazine alone was ingested in the last year in the United States and that much of this was prescribed by physicians other than psychiatrists.

Although most published data concerning these drugs are to be found in psychiatric journals, we must be aware that the drugs are being widely used for emotional problems by non-psychiatrists. It is to them that this paper is directed, to share with them some observations from an intensive study of a few cases in a setting similar to that of their own patients rather than a statistical study of state hospital patients such as already has been done repeatedly. Studies of psychiatric patients in an office setting have not been common.

In the first place, let me state that the much heralded "psychiatric revolution" which was supposed to result from the introduction of these "tranquilizers" or ataractics has settled down to a more calm appraisal and less emotional and more realistic conclusions. These drugs are without question valuable additions to the treatment of anxiety, and it is most probable that new and better drugs will follow, but it must be remembered that there are limitations, dangers, and unpredictable disappointments in the use of the present ones. These dangers can be minimized and the disappointments averted if we keep in mind the limitations this paper will discuss.

Statistical studies indicating percentages of success and failure are misleading and so far are concerned almost exclusively with hospitalized psychiatric patients. The setting of office practice and the types of patient dealt with are so different from state hospital experience that comparison of results is impossible. It is also difficult to define success or failure accurately and to have adequate controls of scientific method.

Because of these difficulties, then, my own statistical results are not significant from the standpoint of accurately defining the value of these drugs. I have, however, given these drugs only to patients in whom there was some reason to anticipate success, and in this selected group one should expect the best results.

In this selected group, a total of 38 patients, 63 per cent had good results and 37 per cent were deemed failures in therapy. These figures are rendered still more conservative by the fact that many of the "good results" group may well have been due to other therapies given simultaneously rather than to the drug alone. Supportive measures, expressive psychotherapy, occupational therapies, and environmental manipulations were employed wherever indicated in addition to the drugs, and it is clear that without such supplementation results would be worse than reported above.

Although ataractics have an important place in management of certain personality disorders, they have limitations and are subject to abuse. The need for further research on such drugs is stressed.

My statistics are given only as a rough indication that many failures occur, and it is with these failures that this paper is primarily concerned. A study has indicated the following classifications of failures:

A. Lack of favorable outcome predicted

1. If depression is the chief or only symptom.
2. When given in insufficient dosage.
3. Where environmental stress is a major factor.
4. When obsessive-compulsive features exist.
5. When a change in basic character structure would be necessary.
6. When these drugs are used as an easy substitute for proper study and diagnosis.
7. Where the drug is used alone instead of as an adjunct to other therapies.
8. To obtain social compliance despite continuance of obvious etiologic factors.
9. Failures due to unknown causes when known factors for success seem satisfied.

B. Adverse effects of the drugs necessitating discontinuance despite temporary favorable outcome or with insufficient trial.

1. Side effects

From the Department of Psychiatry, Topeka Medical Center, Topeka.

Drowsiness, vasomotor instability, muscular weakness.

2. Toxic effects

Jaundice, allergy, agranulocytosis.

A discussion of the above classification will make it more concrete:

1. Many authors have mentioned the worsening of depression with the use of chlorpromazine and reserpine, and attempts have been made more recently to circumvent this by the addition of amphetamines to the prescription. In general where depression is accompanied by psychomotor retardation, feelings of worthlessness, lethargy, disinterest in activities, and such, these drugs are either useless or harmful. But depression of mood with anxiety, agitation, worries, and obsessive thoughts can often be helped by the ataractics. If one keeps in mind that these drugs are valuable in the treatment of *anxiety* and utilizes them primarily for that purpose, then the actual diagnosis or the presence of other symptom complexes should not act as contraindications.

One of our patients, for example, an elderly woman, was so distraught by panic-like anxiety reactions that she became morose, pessimistic, and contemplated suicide. Certainly those were depressive symptoms, but it was felt that anxiety was the chief difficulty and she was placed on large doses of chlorpromazine with great relief. Unfortunately she lived at some distance and was not seen by her family physician for about two months. Her husband had taken the chlorpromazine away from her, and all her original symptoms had recurred. She hanged herself three days later. It is probable that chlorpromazine might actually have prevented suicide in this depressed and anxious woman if it had been continued.

2. Cases of insufficient dosage are commonly seen by the psychiatric consultant. Medical and surgical uses of these drugs, especially chlorpromazine, require relatively small doses, but the relief of massive anxiety sometimes requires huge doses. Since toxic symptoms are not related to total dosage alone, it is generally safe to push dosages to the point where adverse side reactions appear or clinical relief has been accomplished before assuming the drug has failed. I have had some patients on doses as high as 1200 mgs. daily, and doses much higher than this are recorded in the literature. My average dose is much smaller, however, approximately 300 mgs., and I follow all patients carefully for toxic signs, including frequent white blood counts for agranulocytosis.

3. If environmental factors are apparently the major cause of anxiety, it seems that failure of these drugs is more likely. Indeed, even if such potent drugs existed as to tranquilize these patients, their use seems questionable. Let us remember that there are some situations that should be corrected of themselves, and tranquility or equanimity in the face of them would be *abnormal,* especially if it led to acceptance and complacence. In some situations it might be wise to consider that anxiety is acting as a beneficial goad to stimulate action in the direction of changing an adverse situation rather than quietly yielding to it.

4. Obsessive-compulsive states with severe anxiety, phobias, fears of going crazy, and many somatic complaints are sometimes relieved and sometimes not. My experience is about equally divided among successes and failures in such cases, and I know no way of predicting in advance what the response will be. Patients who have seemed to me to be parallel in every way have reacted differently. I suggest that the drugs be used empirically in such cases.

5. Medication cannot be expected to change basic character structure or life-long modes of adapting to stress that are typical of "psychopaths," those with personality disorders, inadequate characters, etc. If symptoms are primarily due to these causes, failures will result. In this group of patients anxiety is rarely felt as such, and it is usually handled by acting out—attempts to alter the environment, or by the expectations of passively foisting themselves on people around them. Since these drugs are chiefly useful in controlling overt, consciously perceived anxiety, their failure to help in such cases can be understood. In fact it is probable that in many such cases they might be contraindicated; they might release what few inhibitions such patients normally possess and lead to further socially unacceptable behavior. It is well known that lobotomy sometimes has such effects, and the use of these drugs sometimes leads to results quite similar to lobotomy.

6. Use of these drugs for convenient and immediate relief of the urgent complaints of patients is common and, of itself, is usually to be commended. If, however, no further attempts are made to discover the causes of the original symptoms, it might be possible to miss situations that need more definitive treatment. As with morphine, no one would practice medicine without it at times, but everyone would be cautious that its use did not mask underlying conditions requiring attention. So with ataractics. We must not be lulled into diagnostic apathy by the absence of complaints following their use. True, we are not always able to find etiological relationships in psychiatric illnesses, and the use of these drugs should not be excluded because of this, but an adequate history, a physical examination with appropriate laboratory studies, and a simple mental status or psychiatric interview should be the minimum. This is not to imply

that every such patient should be seen in psychiatric consultation, but the physician should at least attempt to rule out acute schizophrenic conditions, organic brain disease, drug intoxications, or general metabolic diseases leading to personality changes. Ataractics might even be used in such conditions, but other forms of treatment exist for them and might be preferable.

7. These medications are not to be regarded as the sole form of treatment. For many years before the "ataractic revolution," anxious patients were managed, for the most part quite adequately, by the physician who was willing and able to spend some time in supervising their daily routine, prescribing activities, allowing them to catharse by patient listening, or by more formal psychotherapy. It is my practice to emphasize to all my patients that these drugs are only a supplement to a total prescription of management devices.

8. The continuance of etiological factors which remain uncorrected can be a cause of failure though often we can obtain apparent remissions, changes in behavior, social compliance. We are not sure that all such change is the result of lessened anxiety; sometimes it is the result of a sort of apathetic disinterest, a lobotomy-like effect which is not always desirable.

Frequently patients, and more frequently their families, indicate that relief from anxiety has been marked, but replacing it has been a dull, lethargic, emotionless automaton that isn't human. Symptoms are often the symbolic language of the patient—his way of calling attention to situations that cry out for correction. This is often the case in behavioral problems of children, and merely suppressing these symptoms by pharmacologic means is not good medical practice. Again, because these drugs are sometimes effective in suppressing these symptoms, we may be led to complacency because of their convenience. What such patients need is psychotherapy. Despite its inconveniences, it is directed to underlying problems and not merely to covering them up. There are, of course, many situations that make this treatment of choice impossible and the ataractics are of tremendous value here, but mature judgment is needed to make this decision. Get a psychiatric consultation in such cases.

9. There are still failures that we cannot account for. The complexities of the dynamics of the personality, the physiology of anxiety, and the pharmacology of these drugs must in some unknown ways account for such failures. No doubt in the near future newer drugs will be more uniformly effective, less toxic, more predictable.

In this paper I have not concerned myself with adverse effects of the drugs such as side effects and toxic effects. These have been described elsewhere, and although there no doubt is more to be added to our knowledge of them, I feel I have little of interest to contribute at this time with one exception: muscular weakness, tiredness, lassitude are noticed in many of our patients and do not always seem to be part of a depressive reaction, though patients with depression may complain of these on a psychological basis. We believe these symptoms in many cases are physiological. This seems to be borne out by changing such patients to some of the newer ataractics, some of which seem to control anxiety without causing these side effects.

SUMMARY

1. Ataractics have an important place in the modern management of certain personality disorders, many of which come to the attention of the general practitioner.

2. These new drugs have certain limitations and abuses, some of which have been outlined here.

3. The physician is warned against complacent inattention to etiological and dynamic factors that may be operating and which may be instigated by the convenience and physiological effectiveness of these drugs.

4. Further research will produce more drugs whose physiological effectiveness and pharmacological drawbacks will enhance their convenience and also these dangers.

5. Further study conducted over a period of years is necessary to determine total effects on personality, especially in the developing personalities of children, from these drugs.

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The great problems of the age, international, national and corporate, have to do with the relationships of people. One must be skilled in getting along with others. But this skill must rest upon some such foundation as this: Technical competence; broad intellectual outlook; high sense of honor—moral and spiritual values; attention to the public interest; understanding and appreciation of human relationships.

—Robert N. Hilkert

Iniencephalus

Description of an Abnormal Tubal Human Embryo

PAUL G. ROOFE, Ph.D., ***Lawrence***

This paper has two purposes:

1. To describe an abnormal tubal 3 mm. human embryo, iniencephalus.

2. To request specimens of human embryos of less than a menstrual age of five or six weeks with special notes on their handling.

The abnormal tubal 3 mm. human embryo, iniencephalus, pictured in Figure 2 was obtained in July 1955 by Dr. L. Claire Hays, Hays Hospital, Cedar Vale. A summary of the detailed history is interpreted as follows:

"A normal period was early in May. Conception occurred late in May if we consider the bleeding of June 6-7 as the 'placental sign' which occasionally occurs at about the time of the expected menstrual period. Development then proceeded for from four to five weeks, to judge from the appearance of the embryo. The flow of July 1 appeared after the death of the product of conception. This agrees with the degenerated placenta and the macerated condition of the embryo."

This is a rare specimen of a monster known as iniencephalus. No doubt the ectopic condition contributed to the abnormality. Our knowledge of causes of abnormal growth is limited. Here we see the dorsal flexure of the 18-21 somite period.

Causative factors involved in abnormal development are little known. They undoubtedly can be related to two major areas, heredity and environment. In the present case no doubt the ectopic (tubal) position might be assigned as a reasonable agent for the furrowed occiput and enlarged brain. We cannot assign a blanket causative agent that is responsible for developmental abnormalities. There are definite Mendelian factors operating in such defects as the lack of all or parts of appendages. Another hereditary trait, achondroplasia, is in this category. Cleft palate and cleft lip may readily be Mendelian recessive.

On the environmental side we have very little to go on. Physical factors, such as temperature and radiation, play a major role in producing abnormalities. Temperature, however, may be ruled out in the case of mammalian irregularities of form and structure. However, x-rays and other types of ionizing radiation play a considerable role, especially at the experimental level.

It has been proved experimentally that vitamin deficiency in rats will regularly produce measured abnormal skeletal defects.

Factors influencing abnormalities do so in various stages. One factor may operate in early development and fail to do so in later stages. Some systems are more sensitive than others to these physical and chem-

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The embryo described is of interest because of its rarity. A request is made for additional specimens.

Figure 1. Normal human embryo, 4½ weeks (menstrual age), from Department of Embryology, Carnegie Institution of Washington.

Figure 2. Abnormal human embryo, 4½ weeks (menstrual age), iniencephalus, from Department of Embryology, Carnegie Institution of Washington.

ical factors. Certain inhibiting and accelerating stimuli need not act long. They need to operate only at short intervals during critical periods to produce pronounced abnormalities.

Our aim is not to collect abnormal specimens of embryos or fetuses. What we need and need badly in this area are specimens of human embryos under four or five weeks of menstrual age. Another age group would be fetuses from the third trimester. We use these in many ways in human gross anatomy. Our plastic models of this age group are of considerable value in teaching.

In handling of all specimens it is best, of course, to obtain the material as near as possible to the living state. The specimen should be placed in 10 per cent formalin, and the volume used should be at least 10 times the volume of the specimen. Ship by express, collect, to the Anatomy Department, University of Kansas School of Medicine, Lawrence, Kansas. The container should be absolutely filled to prevent movements of the specimen. If it is packed loosely with absorbent cotton, so much the better. A strict complete case history of the menses and other pertinent data will make possible a scientific understanding of the development of the embryo. This material will be handled discreetly, scientifically, and with care.

Figure 1 is a photograph of a normal 4½-weeks-old human embryo (menstrual age). This permits comparison with the iniencephalus (Figure 2) of similar age.

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The medical profession has been described as being divided as Gaul was, in three parts. One—the active member, always willing to help in every worthwhile action, willing to sacrifice some of his time for the profession or public good. Two—the complacent member, who is theoretically enlisted in the activities of the organized bodies, but actually does nothing more than occasionally attend a meeting. Third—a minority, who simply don't give a damn for anything that does not give them a personal profit.

The profession lives because of the first class. By some form of inciting interest, or even, by compulsory requirements of participation, many, if not most of the second class may be brought into class 1. The third class ought to be eliminated from our efforts and our councils. They are parasites and we will be stronger without them. Why should we hesitate to dismiss them by attendance rules, fair but sternly administered? Those who will not participate, ought not enjoy the benefits available to those who give freely of themselves, their time, and their money. I am not a believer in the desirability of having every medical man in the society. Let it be a privilege gained by effort and participation.

W. U. Kennedy, M.D., President's Page
Journal of the Indiana State Medical Association

PRESIDENT'S PAGE

DEAR DOCTOR:

The emergency session of the House of Delegates was held in Wichita on October 28, and the following action was taken:

(1) The military dependents' care program was approved, designating Blue Shield as the fiscal agent and the medical society as the administrative agent, adopting a fee schedule similar to the Blue Shield $6,000 proposal of a year ago, and authorizing the president to sign a contract in Washington on November 9.

(2) The proposed A.M.A. Code of Ethics, a copy of which was received by all of you, was not approved. Kansas delegates were instructed to do all in their power to defeat that proposal. If they are successful, the Code of Ethics will remain as it now stands.

(3) The House of Delegates unanimously approved amendments to the Basic Science Act and a newly written Healing Arts Act for Kansas.

Another event of significance occurred during the past month —formation of the American Association of Medical Assistants (A.A.MA.). The group became an official organization with the adoption of a constitution and by-laws at a meeting in Milwaukee on October 27. The Kansas Medical Society is proud of the honor bestowed on the state in the election of Miss Maxine Williams of Kansas City as first president of the national group. We are confident that she will perform the duties of her office with distinction. Kansas was well represented at Milwaukee, and it is my hope that more of us will send representatives to the next national meeting, to be held in San Francisco in October of 1957.

Fraternally,

Clyde H. Miller M.D.

President

EDITORIAL COMMENT

Rural Health

The National Conference on Rural Health will be held at Wichita in 1959, as a feature of the centennial celebration of the Kansas Medical Society. The correspondence between the American Medical Association and this office, printed below, tells the story.

September 13, 1956

"Dear Mr. Ebel:

"As Chairman of the Council on Rural Health of the American Medical Association, it is my pleasure to inform you that the Council, meeting in session on September 9, voted unanimously to accept the gracious invitation of The Kansas Medical Society, along with invitations from other leading organizations, and hold its Fourteenth National Conference on Rural Health in Wichita, Kansas, March 5-7, 1959. The Broadview Hotel has been designated as the official headquarters hotel for this meeting.

"Will you please convey to the members of your House of Delegates our appreciation of their consideration and interest.

"In the months ahead we shall look forward to working very closely with you, in order to plan for a most successful National Conference in Wichita.

Cordially yours,
(Signed)
F. S. CROCKETT, M.D., *Chairman*
Council on Rural Health"

October 16, 1956

"Dear Doctor Crockett:

"We are enormously pleased and very deeply challenged by your letter stating that Wichita has been selected for the 1959 National Conference on Rural Health. We shall try our very best to make this meeting completely fulfill all your hopes for such an occasion.

"Clyde W. Miller, M.D., President, has already appointed two committees who will separately and together assist you in the preparations for this event. The chairman of our Committee on Rural Health is V. E. Brown, M.D. of Sabetha. The chairman of the committee planning our Centennial Year is Thomas P. Butcher, M.D. of Emporia. He will, if usual customs prevail, be president of The Kansas Medical Society during that year. We in this office serve as secretaries to both committees and will be pleased to write you from time to time in the near future concerning suggestions these committees might have, or asking questions of you about things the committees might do.

"We are experiencing a feeling of pride over the fact that your National Conference on Rural Health will come to Kansas during our Centennial Celebration and wish to state that the Council of The Kansas Medical Society in formal action taken at Topeka on Sunday, September 30, 1956, authorized the Executive Office to assist you in every possible way in completing local arrangements for the meeting. We in this office are pleased for the opportunity to work with you.

"Again thanks and with best wishes, I remain

Very truly yours,
(Signed)
OLIVER E. EBEL,
Executive Secretary"

Standard Medical Report

The Kansas Medical Society has approved a standard form for reports to insurance companies. After several years' study by the Committee on Medical Economics, a very brief and concise form has been devised. It is modeled after some already in use by other state medical societies where the procedure appears to meet with satisfaction.

For years, physicians have complained because of the time spent in completing form reports. This is augmented because of the fact that many different forms exist and each report is different from the last. Insurance companies are not overly insistent upon their particular report blank. They want the information and appear to be satisfied with standard forms adopted by state medical societies. We, therefore, do not anticipate any problem with insurance companies.

The Kansas approved blank will shortly be mailed with an order blank. Your use of this is strictly optional and not at all required. The Kansas Medical Society is making this available as a personal service to its members, but no one is required to change whatever system he may prefer to use in its stead.

This approved form will give your name and address at the top. In a box it states that the use of this form is approved by the Kansas Medical Society, that the report is supplied without charge, and that additional reports if desired may be had at a cost of $3.00 each.

The body of the report is the center section of one side of a standard size typewriter page. It offers space for identifying the patient, your diagnosis, treatment, prognosis and remarks. On the bottom, below the physician's signature, is a line to be signed by the patient authorizing the report and assigning the benefits if applicable. The paper is light enough to make the preparation of a carbon copy easy.

These standard insurance report forms will be prepared in pads and can be purchased, because of the large volume, at much less than the cost of an individual order. The announcement will be received soon and will carry complete information on cost, etc.

Your Kansas Medical Society believes the use of this form will save the physician's time and is pleased to offer another project of the Committee on Medical Economics as a service to its members.

Leading Causes of Death

The Health Information Foundation, of New York, recently issued a brochure on changes in leading causes of death. The following material is paraphrased from that report.

In 1900 the three leading causes of deaths were communicable diseases, pneumonia-influenza, tuberculosis, and the disease group which includes diarrhea, enteritis, and infective ulceration of the intestines. These diseases accounted for slightly over 31 per cent of all deaths in 1900 but less than 5 per cent in 1954.

The death rate from the pneumonia-influenza category declined 86 per cent; from 12 per cent of the total in 1900 to one per cent in 1954.

The second greatest cause of death in 1900, tuberculosis, declined 94 per cent. Had the 1900 rate prevailed in 1954, there would have been an additional 283,000 deaths from this cause which would have been more than the actual number of deaths from cancer, the second greatest cause of death today.

The death rate from diarrhea and other gastrointestinal conditions has declined 96 per cent since 1900 and by 1940 was no longer in the first ten.

Diphtheria was tenth among the causes of death in the United States in 1900. This rate has declined by 99 per cent since then. Similar declines have occurred in other common communicable diseases.

The major causes of death today are the so-called degenerative diseases and accidents. Heart disease, the leading cause of death today, resulted in over 550,000 deaths in 1954. This is an increase of 129 per cent since 1900. This single cause accounted for 37 per cent of all deaths.

Cancer, with 237,000 deaths in 1954, was second. It accounted for only 4 per cent of the total deaths in 1900 and 16 per cent in 1954 for an over-all increase of 127 per cent.

Vascular lesions, including cerebral hemorrhage, rank third and have remained relatively constant during the past fifty years.

Although accidents rank fourth as a cause of death for the total population, they are the first cause for the ages one through twenty-four years, claiming 34 per cent of all deaths among children one through fourteen years and 52 per cent of the deaths in the ages fifteen through twenty-four years.

The meaning behind these figures and their relationships are, of course, readily understood by physicians. It is felt that the figures and the statistics might be of interest.

Diabetes Week—November 11 to 17, 1956

The American Diabetes Association has set aside this week as National Diabetes Detection Week and is widely publicizing the event. It is suggested that every man, woman and child shall be tested for diabetes during the week of November 11-17, 1956.

The public is being told that one person in seventy-five is a diabetic; that there are two million in the United States of whom one-half do not know they have the ailment. It is estimated that 4,750,000 additional persons living today will develop diabetes during their lifetime, at a rate of 65,000 each year.

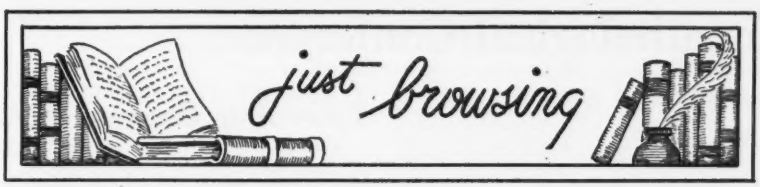
The public is being told that the detection test is simple and takes only a few minutes. They are advised for their own protection and health to have this test taken, and some who heed this advice will come to physicians' offices. The doctor, therefore, should know that this week is Detection Week.

Radioactive Isotope Work in Hutchinson

Grace Hospital and St. Elizabeth's Hospital, Hutchinson, have been approved by the Atomic Energy Commission for radioactive isotope work. The permits for both hospitals allow radioactive iodine uptake studies, Cobalt 60 Vitamin B 12 study for pernicious anemia, use of radioactive iodine for therapy purposes, and use of radioactive phosphorus for leukemia and polycythemia.

A grant of $5,000 for a study of children in rheumatic and non-rheumatic families has been awarded the Department of Medical Microbiology at the University of Kansas School of Medicine by the American Heart Association. This is part of a continuing grant-in-aid approved for three years.

Dr. Tom R. Hamilton is chairman of the department. Dr. Antoni M. Diehl has developed the project with Dr. Wallace Lane, retiring director of the Kansas City-Wyandotte County Health Department, and Dr. Robert W. Weber.



"Cases of disease present, as we say, certain leading symptoms. They thrust forward, like a soldier who presents arms, a complaint such as pain, cough, or 'nervousness,' so that it occupies the foreground of the clinical picture. Such a *'presenting symptom,'* comparable to the *'presenting part'* in obstetrics, may turn out to be of minor importance when we have studied the whole case. But at the outset it has the power to lead us toward right or wrong conclusions in diagnosis, prognosis, and treatment, according as we have or have not learned the art of following it up.

". . . the complaints of the patient—fragmentary expressions of the underlying disease—should be used as *leads,* [which] can be followed to the actual seat of the disease. . . .

"Diagnoses are missed—(a) Usually because physical signs are not recognized; (b) occasionally because we do not think correctly.

". . . correct diagnosis depends upon what enters the doctor's head as possible, and on what his head does to sift the possibilities after they have entered it, as well as on the direct recognition of signs by physical examination.

"To throw open the mind's door and allow *all* disease to enter into consideration each time that we are called to a bedside is foolish in the attempt, and impossible in the performance. Each case should lead us to arrange before the mind's eye a selected group of reasonably probable causes for the symptoms complained of and for the signs discovered. What we select should depend upon the clues furnished us by the patient himself, or by the results of our own examination.

"When, for example, a patient pronounces the word *'headache,'* a group of causes should shoot into the field of attention like the figures on a cash register. Blue lips and finger-nails call up quite another group of ideas. Each clue or combination of clues should come to possess its own set of radiations or 'leadings,' determined partly by what we know of anatomy and physiology, partly by the hard knocks of clinical experience. . . .

"Why do so many practitioners treat symptoms only? Why are their diagnoses and the resulting treatment so full of vagueness, groping, hedging, and 'shot-gun' prescriptions?

"Because they do not know how to get beyond symptoms. They have not been taught from the point of view of practice—i.e., of the presenting symptom. What are the *possible* causes and linkages of any symptom? Which of them are most *probable?* By what methods of questioning or of examination can the *actual* cause be found? . . .

". . . Differential diagnosis [is] a very dangerous topic—dangerous to the reputation of physicians for wisdom. It is, I suppose, owing to this danger that so little has been written on differential diagnosis and so much on *diagnosis* (non-differential). To state the symptoms of typhoid perforation is not difficult. To give a set of rules whereby the conditions which simulate typhoid perforation may be excluded is exceedingly difficult. Physicians are very naturally reticent on such matters, slow to commit their thoughts to paper, and very suspicious of any attempt to tabulate their methods of reasoning.

"Yet all diagnosis must become differential before it can be of any use. All recognition of a lesion or a disease involves distinguishing possible sources of error and excluding them by a reasoning process—more or less definite and conscious. To be of any value, then, diagnosis must descend into the arena where it is questioned and assailed, where all sorts of errors and uncertainties arise to unsettle our wisdom. Those differential tables which we all distrust so much are really no more untrustworthy than the diagnoses we make in practice—for every diagnosis expresses the results obtained by using such a table more or less unconsciously, as we exclude possible errors and alternative diagnoses.

"The attempt to make and defend a differential diagnosis brings all one's failings into sharp relief."

From the introduction and preface to Richard C. Cabot's book, *Differential Diagnosis,* written in 1912.—*O.R.C.*

Clinicopathological Conference

Fever With Progressive, Fatal Coma

CASE PRESENTATION

The case under consideration today is that of a 62-year-old colored male who was admitted to the University of Kansas Medical Center on September 22, 1955, and who expired 30 hours later on September 23, 1955.

The patient was comatose on admission, and the history was obtained from members of his family. During the preceding five years he had been under treatment for hypertension, and for several years he had had a chronic cough which had become worse during the two weeks preceding his admission. Two days before admission he consulted his physician because of double vision and dizziness. At that time his temperature and urinalysis were normal, but the blood pressure was said to be high.

The patient fell out of bed the night before he was admitted to this hospital. He was too weak to return to bed himself, but after being helped back he was rational and talkative. At that time it was found that he was unable to move his right arm and leg. He apparently fell out of bed again the next morning, as he was found in coma on the floor. He was then rushed to this hospital.

The patient had had typhoid fever at 44 years of age. He had had syphilis at age 57 and was treated with penicillin at that time. Sugar had frequently been found in the urine, but the patient refused to go on a diet.

The family history and system review were noncontributory.

On physical examination the patient was found to be an obese colored man who was comatose and who responded only slightly to painful stimuli. The blood pressure was 200/120; pulse 128 and regular; temperature 106.2 degrees rectally; and the respiratory rate was 30. Enlarged painful preauricular lymph nodes were present. The pupils were round and equal and reacted to light. There was focal retinal arteriolar spasm but no papilledema. The neck was somewhat stiff. The chest was clear to auscultation and percussion. It was thought that the heart was enlarged on percussion. The second aortic sound was louder than the second pulmonic sound. There was a grade II systolic murmur present at the apex; it was not transmitted.

The abdomen was soft and obese, and no masses were palpable. The peripheral pulses were good. The genitalia were normal, and the prostate was small and firm. The cranial nerves were intact with the exception of a right central seventh nerve weakness. There was a flaccid paralysis of the right arm and both legs. The left arm was spastic and was held in flexion. Fine clonic movements were visible in the left hand and the left side of the face. The deep tendon reflexes were sluggish in the arms and absent in the legs. There was no clonus, and Hoffman and Babinski signs were absent.

The admission urine had a specific gravity of 1.016, 3 plus albumin, negative sugar, and 8 to 10 pus cells per high power field. There were many hyaline, granular, and bacterial casts in each high power field. The red count was 4,750,000 with 11.2 gm. per cent of hemoglobin. The white count was 17,400 with 85 per cent neutrophils (72 per cent filamented and 13 per cent non-filamented), 11 per cent lymphocytes, 1 per cent basophil, and 3 per cent monocytes. The serologic tests for syphilis were negative. The serum non-protein nitrogen was 55.5 mg. per cent; urea nitrogen, 28 mg. per cent; fasting blood sugar, 175 mg. per cent; cholesterol, 294 mg. per cent; sodium was 133 mEq/L, potassium, 3.3 mEq.; chloride, 94 mEq. and carbon dioxide, 24.4 mEq. Serum amylase was 262 Somogyi units per cent, and serum lipase was 2.0 units per ml.

A lumbar puncture was done; the opening pressure was 170 mm. and the closing pressure 110 mm. There were 78 white blood cells (86 per cent lymphocytes) per cubic millimeter; the sugar was 140 mg. per cent; the chloride, 775 mg. per cent; and the total protein, 42 mg. per cent. The colloidal gold curve was 0111000000. The spinal tap was repeated on the following day with an opening pressure of 280 mm. and a closing pressure of 240 mm. There were 90 white blood cells (82 per cent lymphocytes and 18 per cent polymorphonuclear) and 4 red blood cells per cubic milliliter.

Throughout his hospital stay the patient was comatose except for a few moments when he opened his eyes and spoke briefly. Attempts were made to reduce the high fever with alcohol sponge baths and rectal aspirin, but these were unsuccessful except for two

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Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center as well as by the third and fourth year classes of medical students.

brief occasions when rectal temperature was lowered to 102 degrees. He was treated with 2,000,000 units of aqueous penicillin intramuscularly every six hours and 1.0 gm. of chloromycetin by mouth every 12 hours. He received 200 mg. of cortisone intramuscularly two hours before he died. He was given intravenous fluids for hydration. His urinary output on the day of admission was 800 ml.; it was 500 ml. on the day of death.

Approximately 30 hours after he entered the hospital the patient's respirations became labored, and he died rather suddenly.

Dr. Hugh J. Woods (resident in medicine): I have one addition to the history: it was observed that the patient's preauricular nodes became much larger during his hospital stay, and some observers thought that these were actually the parotid glands.

Dr. Mahlon Delp (moderator): Are there any questions of Dr. Woods?

Kernie Binyon (fourth year medical student):* What was this patient like before he became ill?

Dr. Woods: We know nothing about him except that he was once seen in the outpatient clinic in March, 1955, at which time he complained of pain in his right shoulder. His tendon reflexes were a little hyperactive, and the possibility of his having tabes was mentioned.

Dwight Adams (fourth year medical student): Was a serology done on him?

Dr. Woods: It was negative.

Mr. Adams: What about the urinalysis?

Dr. Woods: There was a slight trace of albumin, no sugar, and rare epithelial and pus cells.

Donald Decker (fourth year medical student): Were there any blood cultures?

Dr. Woods: No.

Mr. Decker: Was there anything unusual about his testicles?

Dr. Woods: No.

Question: Could you give us any more information on the second spinal tap?

Dr. Woods: The fluid was clear, the protein was 52, sugar was 112, and a culture was negative at 72 hours.

Question: Did he have hyperpyrexia during the terminal episode?

Dr. Woods: No, his temperature was about 102 degrees an hour before his death. The medical student said he suddenly stopped breathing, and his heart beat about eight or ten times after that.

Question: Had he lived in Kansas City all of his life?

Dr. Woods: Yes.

Question: Did he have a history of mumps?

Dr. Woods: The history was not clear, but the relatives thought he had never had mumps.

Question: Was only one fasting blood sugar run?

Dr. Woods: Yes.

Question: Was he receiving glucose at that time?

Dr. Woods: No.

Question: Did he have otitis media?

Dr. Woods: No.

Mr. Decker: Would you describe the convulsive movements more clearly?

Dr. Woods: They were intermittent twitching movements of the hands and left side of the face.

Question: Was he in a light or a deep coma most of the time?

Dr. Woods: It was deep.

Dr. Robert Weber (internist): What was his blood pressure during the 30 hours of hospitalization?

Dr. Woods: The systolic pressure was 170 to 200 and the diastolic was 100 to 105 millimeters of mercury.

Dr. Weber: Did it drop at all in the final six hours?

Dr. Woods: Two or three hours before he died his pressure was 180/100.

Dr. Delp: What was his blood pressure when he was in the outpatient clinic in March?

Dr. Woods: It was 230/128.

Dr. Delp: Mr. Boese, will you please present the electrocardiograms?

Kenneth Boese (fourth year medical student): The first electrocardiogram (Figure 1) was taken when the patient was seen in the outpatient clinic in March, 1955, and shows a normal sinus rhythm with a rate of approximately 80. The PQ interval is within normal limits. There is a tall R wave of two and one-half centimeters, and there are inverted T waves in leads I, AVL, V3, V4, V5 and V6. The QRS interval is approximately .08 of a second. This electrocardiogram

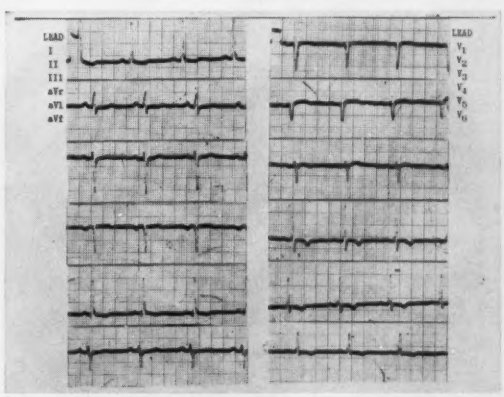

Figure 1

*Though a medical student in March, 1956, when this conference occurred, he, like the others referred to as students, received the M.D. degree in June, 1956.

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[1]Posner, A. C., *et al.;* Further Observations on the Use of Tetracycline Hydrochloride in Prophylaxis and Treatment of Obstetric Infections, *Antibiotics Annual* 1954-55, pp. 594-598.

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shows the pattern of left ventricular hypertrophy, strain, and ischemia.

The second electrocardiogram (Figure 2) was taken the day after admission. The rate is 120. The T waves are inverted in leads I and AVL. The chest leads have reverted back to normal. This tracing shows left ventricular hypertrophy and strain, but there is no ischemia pattern that I can see.

Dr. Delp: Dr. Lin, do you have any comments about the electrocardiograms?

Dr. T. K. Lin (cardiologist): The first tracing showed an interesting sequence from leads four and five, particularly the inverted T waves, which indicated a rather localized ischemia. I saw no definite evidence of left ventricular hypertrophy in spite of the clinical hypertension.

The second tracing showed a sinus tachycardia and a possible ischemic pattern in lead I, but no evidence of ventricular hypertrophy.

Dr. Delp: Mr. Elliott, will you demonstrate the x-rays?

Wade Elliott (fourth year medical student): A shoulder film taken six months before admission shows no bony abnormalities or calcifications in the soft tissues. I interpret this as a normal shoulder.

The cervical spine was x-rayed at that time, and it shows some increased density and lipping of the fifth cervical vertebra and possibly narrowing of the joint space. This could be the result of an old fracture, but it is more likely an osteoarthritic process.

A portable film of the skull taken at the time of admission shows clear frontal sinuses. There is no marked sclerosis of the mastoid process. I interpret this as being a normal skull.

The portable chest film taken on admission, with the patient in some rotation, shows no bony abnormalities and no pathology in the lung fields. I cannot decide about the heart size on the basis of this portable film. The appearance of a mediastinal widening is probably due to rotation. There is a deviation of the trachea which I cannot explain.

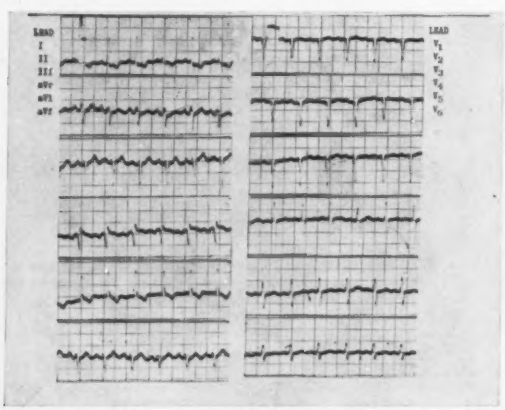

Figure 2

Dr. Delp: Are there any comments, Dr. Germann?

Dr. Donald R. Germann (radiologist): The enlargement of the mediastinum and the tracheal deviation are probably caused by a moderately wide aorta, but I doubt if there is significant tracheal deviation. There is a little left ventricular prominence to the cardiac contour. There is rather marked joint space narrowing of the cervical spine with osteoarthritic spurring around it. It is a degenerative process probably of no importance so far as this admission is concerned.

Dr. Delp: Will you present the differential diagnosis, Mr. Decker?

DIFFERENTIAL DIAGNOSIS

Mr. Decker: We are discussing a 62-year-old man who was admitted to the hospital with the chief complaint of a possible stroke, and who died 30 hours later. There is a history of hypertension and glycosuria. Two days before admission the patient developed double vision and dizziness. He became weak, hemiplegic, and comatose. The physical examination on admission revealed deep coma, hypertension, tachycardia, hyperpyrexia, bilaterally enlarged preauricular lymph nodes or parotid glands, nuchal rigidity, and diffuse neurological signs without papilledema.

The laboratory examinations showed evidence of kidney dysfunction, leukocytosis, elevated serum amylase and lipase, and cerebrospinal fluid abnormalities including an increased cell count (chiefly lymphocytes) and increased pressure. Despite vigorous treatment with antibiotics and cortisone, the patient remained comatose and died 30 hours later.

My differential diagnosis is based on the causes of coma. First, the endogenous and exogenous causes of coma can be quickly ruled out on the basis of laboratory findings, the hospital course, and the clinical findings. Traumatic causes of coma, such as cerebral concussion, cerebral contusion, and acute epidural hematoma can be excluded because there was no history of injury. Subdural hematoma is more difficult to rule out on this basis, but, because of the high fever and equal pupils, I do not think it is the most likely diagnosis.

Cerebral ischemia, such as can be produced by Stokes-Adams attacks and hypertensive encephalopathies, should be mentioned as occasional causes of coma, but we must look elsewhere to explain all the findings that we have in this case.

Primary or metastatic intracranial tumors characteristically present with symptoms of increased intracranial pressure including dizziness, visual signs, vomit-

ing, and severe progressive headache, and the onset can be sudden if hemorrhage occurs into the tumor. Adults frequently present with focal convulsions or other focal signs. The cerebrospinal fluid protein is frequently increased. This case does not fit any of the well outlined clinical tumor syndromes, but one must consider other more diffuse syndromes.

Cerebral vascular accidents must be considered as a cause of coma. Cerebral thrombosis is the most common form of cerebral vascular accident, but consciousness is rarely lost, the patient usually recovers, the cerebrospinal fluid is usually normal, and there is commonly evidence of a local, unilateral lesion. I cannot localize a thrombus which would produce the widespread signs this patient had. Cerebral embolism can be ruled out on the same basis, and there is no logical site for the origin of an embolus.

Subarachnoid hemorrhage usually occurs in younger persons and can be easily ruled out because there was no blood in the cerebrospinal fluid.

Cerebral hemorrhage must be considered because of the patient's hypertension. Characteristically there is sudden onset, coma supervenes, and these patients usually die. There is often a progressive rise in temperature and pulse rate. The cerebrospinal fluid may be under increased pressure, and there may be increased protein and pleocytosis. In about 50 per cent of such patients the cerebrospinal fluid is bloody, but that is not diagnostic. Bleeding into a ventricle can also produce hyperpyrexia. I ruled out cerebral hemorrhage because I cannot localize a vascular lesion which would produce such widespread bilateral neurological deficit without producing more cranial nerve signs.

Bilateral carotid artery thrombosis usually occurs in patients who have a history compatible with thrombosis of one side. Blindness is frequently produced, and the cerebrospinal fluid is normal. The high fever in this case speaks against this.

Cerebral venous lesions such as lateral sinus thrombosis and superior sagittal sinus thrombosis must also be considered. They are preceded by a bacterial infection such as mastoiditis or paranasal sinusitis, but the infection may not be recognized. Typically there is a septic fever rather than a sustained high fever such as this patient had.

The septic causes of coma must be strongly considered in this patient. The bacterial meningitides can quickly be ruled out on the basis of cerebrospinal fluid findings. They produce a greatly increased polymorphonuclear count and depress the glucose, and the organism can usually be cultured from the cerebrospinal fluid. Brain abscess usually has a slow onset. The degree of pyrexia that this patient had would be unusual unless the abscess ruptured into the subarachnoid space or into the ventricles. I do not think that this is the most likely diagnosis.

Central nervous system syphilis and syphilitic gummas can be ruled out on the basis of the negative serology.

The mycotic encephalitides characteristically produce a chronic illness and cerebrospinal fluid findings which are not like those in this case. They elevate protein and depress the sugar.

The rickettsial encephalitides are nearly always accompanied by a skin rash, and they are very rare.

All of the findings in this case are compatible with a viral meningo-encephalitis. St. Louis encephalitis may have a sudden onset with diffuse cerebral and meningeal signs, acute rise in temperature up to 106 degrees, progressive coma, and a mild pleocytosis, especially of the mononuclear cells. The rest of the cerebrospinal fluid findings are compatible with St. Louis encephalitis. Convulsions may occur and mortality is high, particularly in the older age groups. Western and eastern equine encephalitis are capable of producing this same clinical picture, but they are rare in this region.

The possibility of mumps encephalitis should not be dismissed lightly, although it is uncommon in this age group, and fatal cases are extremely rare. Donahue[1] reported 20 proved cases. The bilateral preauricular swellings might have been the parotid glands, and the elevated serum lipase and amylase would be compatible with mumps.

One should mention other secondary meningoencephalitides such as those caused by measles and mononucleosis, but they are rare in this age group.

In summary, the best diagnosis in this case is viral meningo-encephalitis. I would only be speculating to pin it down to any particular etiological agent. I cannot, with certainty, rule out cerebral hemorrhage, hemorrhage into a brain abscess, hemorrhage into a brain tumor, or bilateral carotid artery thrombosis.

CLINICAL DISCUSSION

Dr. Delp: What is your final diagnosis?

Mr. Decker: Meningo-encephalitis, probably viral. If I had to pin it down, I would say St. Louis encephalitis.

Dr. Delp: Mr. Binyon, did you rule out mumps?

Mr. Binyon: Not entirely.

Dr. Delp: Do you think that it probably was mumps?

Mr. Binyon: My diagnosis is a meningo-encephalitis of viral etiology. I do not know whether or not it was mumps.

Dr. Delp: Mr. Deosaransingh?

Manohar Deosaransingh (fourth year medical student): I think that he had meningo-encephalitis,

but I do not know the cause. It is more or less an arbitrary diagnosis, and I doubt very much whether the pathologists can get any closer to the etiology than we can.

Dr. Delp: Mr. Doubek, did you rule out a vascular lesion, and if so, how?

Herbert Doubek (fourth year medical student): I did not. The onset with the patient falling out of bed goes along with a vascular accident and progressive coma, but I think that the spinal fluid findings are against it.

Dr. Delp: Mr. Elliott?

Mr. Elliott: I agree with the diagnosis of viral meningo-encephalitis, but I am a bit disturbed by the sugar falling in the second spinal tap. I do not expect the sugar to go down in the spinal fluid without a correlated blood sugar fall.

Dr. Delp: Mr. Decker, how did you deal with that problem?

Mr. Decker: I did not deal with it.

Dr. Delp: How would you explain the patient's fasting blood sugar of 175 mg. per cent?

Mr. Decker: I think he had diabetes mellitus.

Dr. Delp: Do you think that this contributed to his death?

Mr. Decker: I think that he would have died anyway.

Dr. Delp: Dr. Berry, there have been frequent references here to St. Louis encephalitis. I want to call on someone who lived through the epidemic of Kansas City encephalitis. Do you think that this picture at all simulates your experience with patients in that epidemic?

Dr. Maxwell G. Berry (internist): Equine encephalitis is a possibility if this family lived outside the city limits or close to a reservoir of some kind. As most of you know, the main reservoir in rural Kansas and Missouri is the horses. The onset in September suggests that he may have been bitten by a mosquito. The encephalitis that I have seen in Kansas City itself has been sporadic cases of an encephalitis which is caused by an unknown virus.

A year ago I saw a few patients in Colby who had equine encephalitis. The reservoir was right there in a horse barn. The mosquitoes were in the backyard, and they were Anopheles, too. We had a little trouble getting the public health service to work on it, but when they did they traced it down very well. To make a diagnosis of encephalitis in this patient, we have to overlook a lot of things. You have more red herrings in this protocol than any I have seen for a long time, i.e. the long history of hypertension, the hyperglycemia, and a colored man with a chronic cough. These situations are quite suggestive of widely different diagnoses. Those things are a little bit difficult to overlook. If he did have encephalitis, it would have been more convincingly suggested by his living in the outskirts of town where mosquitoes are more plentiful.

Dr. Delp: Dr. Weber, what was your diagnosis?

Dr. Weber: I saw this patient, and I made the diagnosis of mumps meningo-encephalitis.

Dr. Delp: You thought the man had parotitis?

Dr. Weber: Yes, and I thought that the blood sugar might have been related to pancreatitis.

Dr. Delp: Do you think the amylase values are compatible with that?

Dr. Weber: They are not high enough for an acute pancreatitis.

Dr. Delp: Did he have any abdominal tenderness when you examined him?

Dr. Weber: I could not elicit anything, but it was two or three hours before he expired.

Dr. Delp: I doubt if he had any abdominal tenderness. I did not examine him personally, but I watched several others examine him.

PATHOLOGICAL REPORT

Dr. Frank A. Mantz (pathologist): External examination of the body disclosed that the individual was moderately obese, and there was bilateral enlargement and swelling in the preaũricular area.

The parotid glands were enlarged, exceedingly soft, and of a distinct yellow color. They show the picture of fat replacement which is not uncommon in people who are obese and in older persons. Similar fat replacement was found in the pancreas. No evidence of inflammation was found in the gland.

A general systemic examination yielded little of particular note. There were few, if any, stigmata of diabetes. A mild degree of arteriosclerosis was detected.

The heart weighed 680 grams and showed marked left ventricular enlargement. No valvular lesions were detected, and the coronary vessels were patent throughout. The kidneys, although not significantly con-

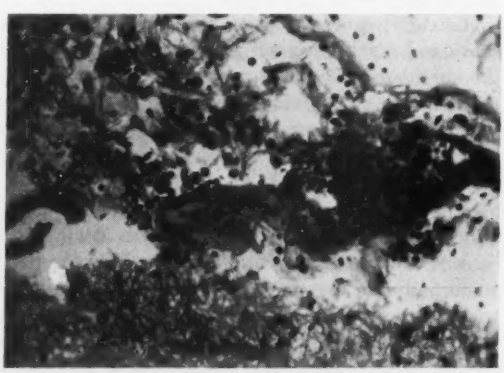

Figure 3. Right parietal leptomeninges showing inflammation with edema and predominantly lymphocytic exudate.

tracted, showed fine granularity, and on microscopic examination there was a significant degree of arteriolosclerosis associated with mild thickening of the basement membrane of the glomerular capillaries.

The gross examination of the brain showed no defect. The brain weighed 1340 grams, was not significantly hyperemic, and showed no evidence of increased intercranial pressure; on section no discrete lesions were encountered. Histologically, however, characteristic findings were noted. There was a diffuse mild meningitis manifested here by thickening and edema of the meninges and by a moderate infiltration of leukocytes, mostly lymphocytes (Figure 3). The meningitis was rather patchy in distribution. It was seen most intensely over the frontal and parietal regions and to a lesser degree over the base. No meningitis was detected over the cerebellum, about the brain stem, or about the cervical cord. The ependyma was not involved, and there was no infiltration in the choroid plexus.

The next characteristic lesion was in the neuron, which displayed a moderate degree of what might be classified as acute cell disease. This is manifested here by loss of Nissl's substance, swelling and smudginess of the nuclei (chromatolysis), and moderate satellitosis by glial cells adjacent to the neurons (Figure 4). In some areas frank necroses of the neurons had occurred, producing tiny glial reaction sites. In the cortex there were, in addition, clusters of glial elements, mostly microglia, unassociated with degenerating neurons, following the tiny capillaries (Figure 5). These tiny glial nodules were the most frequently encountered and probably the most significant change in the brain substance.

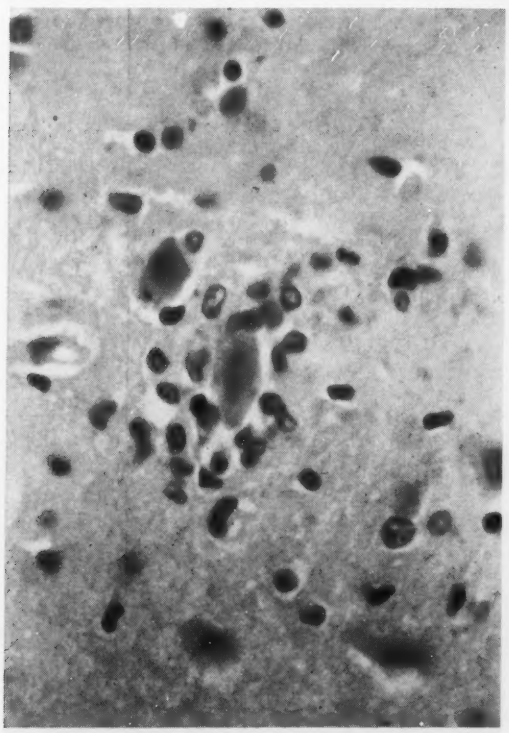

Figure 4. Right parietal cortex showing neuronal necrosis and satellitosis.

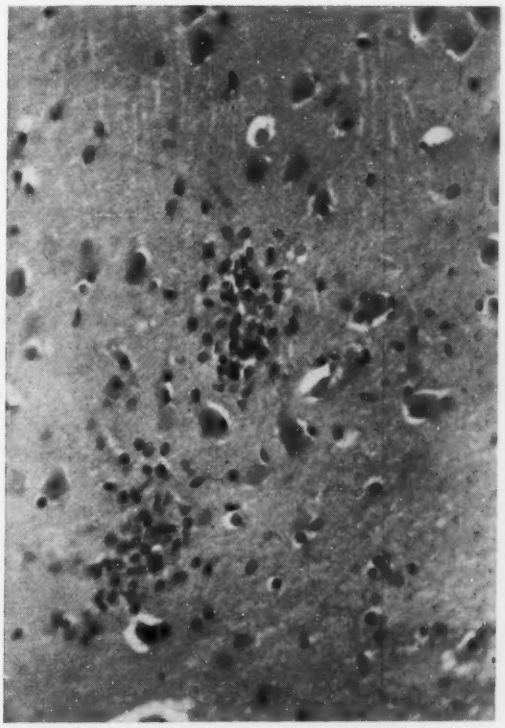

Figure 5. Hippocampal gray matter showing microglial nodules.

A third and striking lesion that was present throughout the brain was perivascular cuffing with lymphocytes in which the Virchow's space about the blood vessels was literally stuffed with lymphocytes (Figure 6). Changes within the brain were mainly within the basal ganglia, the midbrain, the pons, and the medulla.

A section from the caudate nucleus showed what might be classified as a fourth type of lesion, that of focal parenchymal degeneration unassociated with significant neuronal damage (Figure 7). In such sites there is some evidence of demyelinization, but characteristic perivascular demyelinization is not seen in this case. The cells which compose the reaction here are largely microglia. The globus pallidus was similarly involved. In fibers which traverse the internal capsule one encounters occasional small lesions accounting

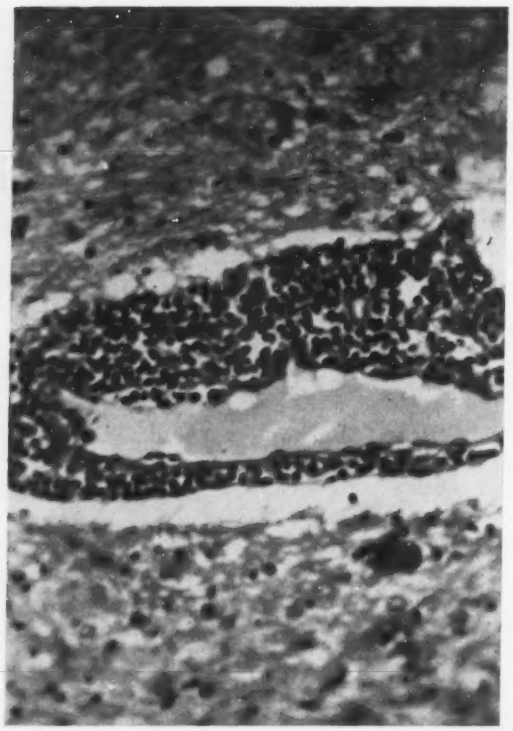

Figure 6. Midbrain in region of substantia nigra showing marked perivascular lymphocytic infiltrate in Virchow Robins space.

for whatever upper motor neuron changes this patient may have had.

The most marked changes were encountered within the substantia nigra which bilaterally was diffusely involved with wide areas of parenchymal degenerative changes. The brain stem likewise was affected, the involvement extending throughout the reticular substance.

These findings are classical of a viral type encephalitis. There is diffuse distribution throughout the brain substance, which suggests that this was an encephalitis that had been blood-borne. The absence of involvement of the ependyma suggests that lymphocytic chorio-meningitis is not a factor here. Our failure to demonstrate intracellular inclusions excludes rabies, herpes simplex, and inclusion encephalitis. This probably belongs to a large group of insect-borne viral diseases which include Japanese B encephalitis, Australian X, California, and a number of other somewhat esoteric diseases which can be excluded on the basis of geography. That leaves us with St. Louis encephalitis, eastern equine encephalitis, and western equine encephalitis. Mumps encephalitis cannot absolutely be ruled out, but one would expect to find considerably more perivascular demyelinization than one sees here. The pathologist finds himself at an impasse and must enlist the aid of a virologist to determine the definitive diagnosis. For this reason I would like to ask Dr. Werder to relate to you the studies that he conducted on material which we gave him.

Dr. Alvar A. Werder (microbiologist): When we think of the eastern and western equine viruses, we should remember something about where they can be found and what their natural hosts appear to be. We have known for about 20 or 25 years that they can be pathogenic for man. St. Louis encephalitis has been common in this area as well as in St. Louis. Western equine, as the name implies, is common in the western part of the country, but it is spreading eastward all the time. Eastern equine encephalitis is mainly found in the eastern part of the country, but it has also appeared in Texas and Arkansas and is moving westward. It has not been reported in Kansas.

We no longer believe that the horse is the natural host. Chickens, pigeons, wild fowl, and some wild animals are now thought to be the natural host. The virus is spread accidentally by bite of either Anopheles or Culex mosquitoes, mites, or ticks to horses, mules, and human beings.

It was surprising to us that we did find evidence

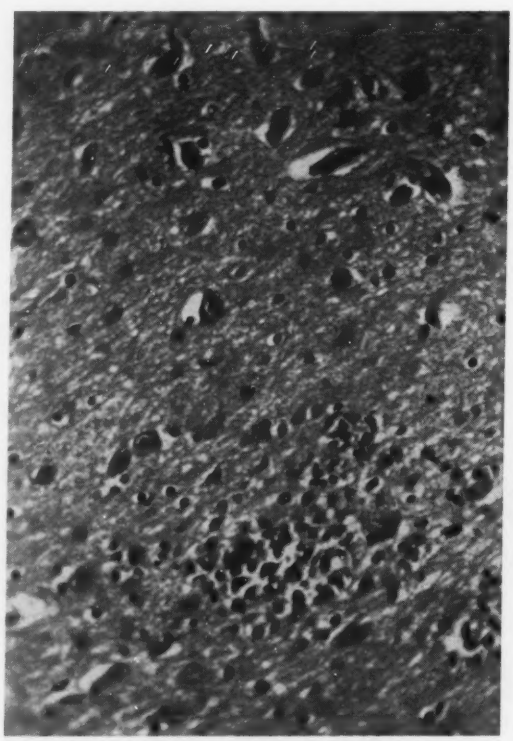

Figure 7. Caudate nucleus showing focal parenchymal degeneration with mild glial reaction.

that pointed to eastern equine encephalitis virus. We inoculated brain tissue into young mice. It caused an illness characterized by ruffled fur, hunching of the back, paralysis, and death. This could be neutralized by the addition of serum from patients who had convalesced from eastern equine encephalitis, but it was not neutralized by serum from patients who had had St. Louis or western equine encephalitis. This evidence points strongly to the fact that our patient had eastern equine encephalitis.

Dr. Delp: Thank you, Dr. Mantz and Dr. Werder. Once more we have a case at this session in which death was due to an infectious process, something I always like to emphasize. I was not being facetious when I mentioned Kansas City encephalitis. There was an epidemic of encephalitis in St. Louis and Kansas City in 1933, 1934, and 1935. Although St. Louis received most of the publicity, and the virus has been named after that city, some investigators have felt that there was a subtle difference between the two epidemics and have occasionally referred to the epidemic here as Kansas City encephalitis. I would not have guessed that this was eastern equine encephalitis because my own impression has been that it usually occurs in children, seldom in adults. Western equine encephalitis is essentially a disease of adults.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Primary

Eastern equine encephalitis, advanced (history of diplopia, fever, and coma developing progressively over three days prior to death and post mortem isolation of the virus with identification through neutralization tests).

Puncture wounds in the lumbar region of the back (history of spinal punctures revealing increased cerebrospinal fluid pressure, 90 leukocytes per cubic millimeter with 82 per cent lymphocytes, total protein 52 mgm. per cent, sugar 140 mgm. per cent, and chloride 775 mgm. per cent).

Hyperplasia of bone marrow, moderate (history of leukocytosis, 17,400 per cubic millimeter with 82 per cent polymorphonuclear neutrophil leukocytes).

Depletion of cortical lipid of adrenal glands.

Fatty metamorphosis of the liver, slight.

Accessory

Arteriosclerosis of the aorta and coronary arteries, moderate, and of the cerebral, pulmonary and celiac arteries, slight.

Hypertrophy and dilatation of the heart, 620 gm. (history of hypertension with blood pressure 230/130 for undetermined period).

Arterio and arteriolonephrosclerosis, slight.

Emphysema of both lungs, slight.

Calcified nodule, tracheobronchial lymph node.

Obesity, moderate, weight 215 pounds.

Fat replacement of the parotid glands, advanced.

Fat infiltration of the myocardium and pancreas, slight.

Small submucosal neurofibroma of the ileum.

Small hemangioma of the liver.

Focal calcification of renal tubules, slight.

Small retention cyst of both kidneys, slight.

Nodular hyperplasia of the prostate, slight.

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PHYSICIANS' ACTIVITIES

Dr. Jerome S. Menaker, Wichita, was invited to serve as a speaker at a meeting of the American Academy of Obstetrics and Gynecology in Chicago early this month.

Dr. David Robinson, professor of surgery at the University of Kansas School of Medicine, participated in a symposium on surgery of the eyelids at a meeting of the American College of Surgeons in San Francisco early last month. Later he conducted a course on surgery of irradiated tissue for the American Society of Plastic and Reconstructive Surgeons at Miami Beach.

Wyandotte County has made plans for providing a full time resident physician for the county home and hospital and has appointed **Dr. Lennert B. Mellott,** Bonner Springs, to that position.

"Skin Cancer" was the subject of an address given by **Dr. A. E. Hiebert,** Wichita, before a recent meeting of the Sedgwick County Chapter of the American Cancer Society.

Dr. LaVerne B. Spake, Kansas City, was notified recently that his portrait will be added to a collection of photographs of leaders in the medical profession in the armed forces medical library in Washington.

Dr. A. R. Cuadrado, Sharon Springs, has announced plans to open an office in Goodland and practice there four half-days a week while continuing to practice in Sharon Springs the remainder of the time.

Dr. Rodger A. Moon and **Dr. C. Arden Miller,** of the University of Kansas Medical Center, presented a case of psychiatric disturbance in a preschool child at the annual meeting of the Kansas Council for Children and Youth in Emporia last month.

A three-year residency in anesthesiology at the Mayo Clinic, Rochester, has been begun by **Dr. Bernard A. Brungardt,** Salina.

Dr. Robert G. Loudon of Edinburgh, Scotland, who recently completed a course in pulmonary diseases at Brompton Hospital, London, arrived in Kansas last month to join the medical staff of the new Southeast Kansas Tuberculosis Hospital in Chanute.

Dr. Howard E. Snyder, Winfield, was nominated as a member of the board of the American Cancer Society at a meeting of Region 7 at Little Rock recently. **Dr. J. P. Berger,** Wichita, was elected chairman of Region 7, which includes Arkansas, Kansas, Missouri, Oklahoma, and Texas.

Dr. William L. Valk, of the University of Kansas Medical Center, is serving as a member of the A.M.A. Residency Review Committee. He has inspected residency facilities in California and Louisiana.

Dr. Robert E. Bolinger, associate professor of medicine at the University of Kansas School of Medicine, is on sabbatical leave, August 1, 1956, to July 1, 1957, to study insulin and metabolic diseases in Holland. The greater part of the time will be spent at the University of Amsterdam.

Dr. D. V. Conwell, Wichita, has been appointed a member of the Committee on Preliminary Arrangements for the second Pan-American Congress of Gerontology, to be held in 1958.

Dr. Donald L. Rose, Kansas City, was named president-elect of the American Congress of Physical Medicine and Rehabilitation at a meeting held at Atlantic City in September.

Dr. Victor E. Chesky, Halstead, became a fellow of the International College of Surgeons at a meeting held in Chicago last month.

Dr. J. O. Osborne, formerly of Prairie Village and Kansas City, has opened an office in Eudora.

Dr. William J. Reals, Wichita, is one of three authors of an article, "Determination of Protein-Bound Iodine by the Leffler Method," published in the September issue of *U. S. Armed Forces Medical Journal.*

"So You Have an Allergy" was the subject of a talk given by **Dr. Harry Lazar,** Wichita, before a recent meeting of the Sedgwick County Medical Assistants' Society.

A retired physician, **Dr. Jacob H. Enns,** Newton, has enrolled as a non-credit student in the University of Wichita's first political science telecourse. Dr. Enns will observe the 50th anniversary of his graduation from Fairmount College, now the University of Wichita, next June.

Dr. Harold W. Houk, an Oklahoman who recently completed internship in Wichita, opened an office in Wellington on October 1.

A feature story about **Dr. Ralph R. Melton,** Marion, who has made a hobby of studying Kansas history, was published in a recent issue of the *Wichita Eagle.* With his family Dr. Melton has collected a vast amount of historical data which is being preserved in the Marion City Library and the Marion County courthouse.

Dr. R. E. Riederer, Olathe, addressed the Washington School Parent-Teacher Association there recently on the subject of the school health program.

Dr. H. Preston Palmer, Scott City, recently celebrated his 25th anniversary in practice there. A story about him was published in the *Scott City News Chronicle* at the time, telling of changes in medical practice during the 25-year period.

"Health Facts and Fallacies" was the subject discussed by **Dr. Christine Thelen,** Wichita, at a recent meeting of dental assistants in Wichita.

The establishment of the **Dr. F. D. Lose** and Ann Lose medical student loan fund at the University of Kansas School of Medicine has been announced. Dr. Lose, who was a member of the first graduating class of the school, has practiced in Madison for 50 years, and the community expressed its appreciation by contributing $1,500 to the fund.

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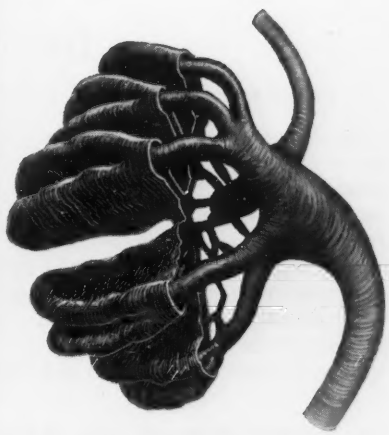

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SEARLE

Dr. Harold L. Graber, formerly of Hutchinson, has moved to California and has established his practice in Fullerton.

Dr. Jesse R. Pritchard, Fort Scott, has been named health officer of Bourbon County, replacing Dr. Ralph Y. Strohm who has resigned after 10 years of service.

A feature story about **Dr. Etta Mundell,** retired Hutchinson physician, was published in the *Hutchinson News-Herald* recently as part of the observance of National Businesswomen's Week.

Dr. Edward X. Crowley, Wichita, became a fellow of the American College of Surgeons at a meeting in San Francisco on October 12.

Dr. Edward J. Fitzgerald, formerly of Columbus, Nebraska, has moved to Wichita to become director of the department of radiology at Wichita-St. Joseph Hospital.

The Union Pacific Railroad has appointed **Dr. A. R. Cuadrado,** Sharon Springs, as its physician in the Sharon Springs area.

DEATH NOTICES

Ione Schultz Clayton, M.D.

Dr. Ione S. Clayton, 71, an honorary member of the Cowley County Society since her retirement in 1953, died at her home in Arkansas City on September 16. She was graduated from the University of Illinois College of Medicine in 1908 and came to Kansas the following year, continuing in practice in Arkansas City until retirement. She had served locally as a member of the board of education and statewide as a member of the board of health.

Samuel Glick Ashley, M.D.

Dr. S. G. Ashley, 74, who had practiced in Neosho County since 1908, died in Chanute on September 17, two weeks after suffering a cerebral hemorrhage. He was an active member of the Neosho County Society. He was a graduate of the University Medical College of Kansas City, class of 1908, and practiced first in Earlton, moving to Chanute in 1924. In recent years two sons, Dr. Ed Ashley and Dr. Glen Ashley, had been associated with him in practice at the Ashley Clinic.

Albert Newton Gray, M.D.

An 86-year-old physician who remained active in practice until 10 days before his death on September 20 was Dr. Albert Newton Gray, Burlington. He had practiced in Burlington since 1917, having practiced first in central Kansas and Utah after his graduation from the University of Kansas School of Medicine in 1907. At the time of his death he was an honorary member of the Coffey County Society, an organization he served as president. He was also health officer of Coffey County.

George William Bertram Beverley, M.D.

A veteran physician, Dr. G. W. B. Beverley, 84, died at his home in Topeka on September 24. He was an honorary member of the Shawnee County Society. Dr. Beverley received part of his education at the University of Bristol in England, receiving his degree from that school in 1897. He later studied at McGill University in Montreal, Canada, and received his medical degree from University Medical College of Kansas City in 1903. He began practice in Alma and left there to serve in the Army in World War I. He opened an office in Topeka when he returned from military service, and he remained in practice there until his retirement in 1942.

Perry Marshall Bell, M.D.

A Wichita physician who had served as Sedgwick County physician for 26 years, Dr. P. M. Bell, 67, died at a Wichita hospital on October 3 after a short illness. He was graduated from Howard University College of Medicine, Washington, in 1914 and began practice in Wichita after internship in Washington and a residency in Kansas City, Missouri. He was an active member of the Sedgwick County Society.

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Dr. Donald L. Rose, Dr. Stanley F. Radzyminski, and **Dr. Ralph E. Beatty,** of the University of Kansas School of Medicine, received the gold medal of the American Congress of Physical Medicine and Rehabilitation at the organization's September meeting in Atlantic City. It was awarded for their exhibit, "Effectiveness of Brief Maximal Exercise on the Strength of the Quadriceps Femoris."

COUNTY SOCIETIES

Members of the Shawnee County Society were hosts at a meeting of the Golden Belt Medical Society held at the Hotel Jayhawk, Topeka, on October 4. Dr. William Spanos of Kansas City spoke on "Toxemias of Pregnancy," and Dr. Derek Miller, Topeka, discussed "The Tranquilizing Drugs."

Dr. Arthur Grollman, chairman of the Department of Experimental Medicine, Southwestern Medical School, was guest speaker at a meeting of the Sedgwick County Society on October 2. He discussed "Endocrine Disorders in Everyday Practice."

A meeting of the Southeast Kansas Medical Society was held at the Columbus Country Club last month with members of the Cherokee County Society as hosts. Dr. Kenneth E. Cox, Kansas City, Missouri, was guest speaker. At the business session Dr. William T. Braun, Pittsburg, was named president, Dr. Raymond W. Lance of Arma was elected vice-president, and Dr. Dick B. McKee, Pittsburg, was chosen as secretary-treasurer.

Two speakers presented a program at a meeting of the Shawnee County Society held at Topeka on October 1. Mr. Otto Schnellbacher gave a review of the insurance program open to physicians, and Capt. C. P. Hungate, M.C., U.S.N.R., Olathe, spoke on "Problems in Management of Atomic Blast Injuries." At the business session Dr. Harry J. Davis was elected to honorary status and the following were elected to active status: Doctors Lawrence M. Agan, M. Kirk Miller, Jr., Charles F. Pierce, William R. Powell, Oscar S. Proctor, and Bartlett W. Ramsey, all of Topeka.

A meeting of the Wyandotte County Society was held at Kansas City on October 16. Two guests, Mr. David Morantz and Mr. Claude E. Housman, discussed "The Problem of Collecting Delinquent Accounts in Medical Offices."

A program on thyroid diseases was presented at the September meeting of the Riley County Society held in Manhattan. Speakers were Dr. Albert E. Upsher, pathologist; Dr. Ward Wilhelm, internist, and Dr. Sam Chapman, radiologist, all of Kansas City.

Surgeons Elect Officers

Officers for the coming year were elected by the Kansas Chapter of the American College of Surgeons at a meeting held at Newton recently. Dr. John A. Grove, Newton, was named president; Dr. Orville R. Clark, Topeka, president-elect; Dr. Robert W. Myers, Newton, secretary-treasurer; Dr. Alfred G. Dietrich, Newton, and Dr. William P. Williamson, Kansas City, councilors.

The scientific program for the day included papers by Dr. Grove, Dr. John G. Shellito of Wichita, Dr. Robert E. Pfuetze of Topeka, and Dr. Stanley L. VanderVelde of Emporia. Dr. Clark was moderator for a panel discussion presented by Dr. Dietrich, Dr. Jack Graves of Wichita, and Dr. Wendell A. Grosjean of Winfield.

Grant for Rheumatic Fever Study

A grant of $5,500 for a year's study of rheumatic fever in families has been made by the American Heart Association to Dr. Tom R. Hamilton, chairman of the Department of Microbiology at the University of Kansas School of Medicine. The study was begun 21 months ago under a previous grant.

Fifty families with members who have had rheumatic fever have been studied. The use of long-lasting penicillin to prevent throat infections that precede rheumatic attacks was successful, the study revealed. In the next 12 months the program will study "matched" groups of families. Comparisons will be made of families with present cases, those with past cases, and those with no cases in their history.

Society View of Prepayment Plans

The Wayne County (Michigan) Medical Society recently authorized publication of the results of a study made by its Committee on Prepaid Medical Care Plans. The report included the following points: (1) The public wants a medical care plan which provides complete coverage; (2) The medical profession should assume leadership in determining the type and form of prepaid medical plans; (3) A prepaid medical care plan should embody a sense of responsibility on the part of the profession, but just as importantly a sense of responsibility on the part of the patient, and (4) The prepaid medical care plan should preserve the traditional right of the patient to select a physician of his own choosing.

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THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

In addition to helping states make monthly public assistance payments to certain indigent persons, the federal government for a number of years also has contributed to the cost of their medical care. Because the grants formula is somewhat complicated, and the amount of medical care varies with the states, this U. S. contribution cannot be fixed definitely. It is estimated at about 90 million dollars a year.

About a third of the states now deposit these federal grants—which must be matched 50-50—in a separate fund, from which the medical care costs are paid directly to the vendors, such as physicians, dentists, hospitals, nursing homes, and druggists. The remaining two-thirds include medical care costs in monthly checks to the indigent and expect these people to pay their own medical bills.

But beginning next July 1, this U. S.-state medical care arrangement is going to be drastically altered.

For one thing, the U. S. will increase its payments from the current $90 million a year to between $200 million and $300 million. For another, *all* medical care money under the new program will be put into a separate fund, from which the indigents' medical bills will be paid, in one way or another, by the state itself.

It is true that in some states the new program will not have much effect. This will be the case with those states that already have a substantial medical care program and see no reason for increasing it and with those unable to raise the matching money.

But the amount of money potentially available to each state is significant, and in most states the changeover from the old to the new systems will have an important effect on physicians and other vendors of medical care. For example, eight states will have "new" medical care funds in excess of 10 million dollars, if they put up half the money. California's potential fund is $27 million and New York's and Texas' more than $18 million each.

Before state welfare directors can start operating under the new program, they will have to decide (a) whether they will require doctors to agree to a fee schedule, if one is not already in operation in their indigent care program, and (b) how the doctors will be reimbursed (whether through their societies or other mechanisms, or directly by the government). Some state welfare officials already have approached state medical societies to talk over the situation.

(U. S. contributes to indigents in only four categories—the aged, dependent children, the blind, and the disabled. For their medical care, it will offer states $3 per month for each adult and $1.50 for each child, money which the state must match. It is out of these funds that payments will be made for medical care.)

NOTES

Because most applicants did not supply enough information, the council in charge of grants for medical research facilities approved only a handful of projects at its first meeting. Although $30 million was available, only $764,159 was allocated. Money went to seven institutions. However, the expectation is that the fund will be just about exhausted at the December meeting of the council, as more than 250 hospitals, schools, and laboratories have asked for money.

First head of the new National Library of Medicine is the man who steered the Armed Forces Medical Library through the last seven troubled years —Col. Frank B. Rogers. He is on loan to PHS, which is in charge of the new institution to be built up around AFML.

Hearings will probably be held in December by the House Interstate and Foreign Commerce committee on federal aid to medical education. The expert panel system will be used, instead of lone witnesses. Currently the committee staff is analyzing information received in response to questionnaires sent out to about 60 organizations interested in medical education.

A six-man advisory committee, named by Secretary Folsom, is attempting to work up suggestions that will help hospitals improve care and reduce costs. Some possibilities: central cafeterias for ambulatory patients, light housekeeping work done by some patients themselves.

Regional Small Business Administration offices now are taking applications for loans to three types of health facilities—hospitals, nursing homes, and medical and dental laboratories. Institutions must be "small" and must be run for private profit.

Real intelligence is a creative use of knowledge, not merely an accumulation of facts. The slow thinker who can finally come up with an idea of his own is more important to the world than a walking encyclopedia who hasn't learned how to use information productively.—*D. Kenneth Winebrenner.*

Silence is a talent as greatly to be cherished as that other asset, the gift of speech.—*Josephine Lawrence.*

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Myocardial Infarction

Study of Syndromes Related to Inadequate Oxygen Supply of the Myocardium

BRUCE JOSEPH, M.D., ***Honolulu***

Angina pectoris, acute coronary insufficiency, and myocardial infarction are terms which have been applied clinically to three syndromes related to inadequate oxygen supply of the myocardium. Angina pectoris is characterized by anterior chest pain occurring with exercise or emotion or after eating but subsiding with rest in two or three minutes. Myocardial infarction is characterized by severe, crushing chest pain of long duration, associated later with signs of tissue death such as an increased sedimentation rate, fever, and leukocytosis. Intermediate between these two conditions is acute coronary insufficiency, with pain lasting 15 to 30 minutes but without clinical evidence of tissue necrosis. Of course, all degrees of myocardial ischemia occur, producing clinical features varying from one extreme to the other. For this reason the division into three groups, although of clinical significance, is arbitrary.

Relative myocardial ischemia may occur either when there is an increased demand of the myocardium for oxygen, as in tachycardia, cardiac hypertrophy, hypertension, valvular disease, and muscular exercise, or a decreased oxygen supply to the heart. The latter may be the result of anemia, diffuse chronic pulmonary disease, or decreased coronary blood flow.

Since the coronary blood flow takes place during diastole, and is directly proportional to diastolic blood pressure, a lowered diastolic pressure from shock or aortic insufficiency may decrease coronary flow considerably. Thus, rheumatic heart disease can not only increase the work of the heart through valvular malfunction but may also decrease coronary flow if aortic insufficiency is present. Syphilitic aortitis may result in the narrowing or occlusion of the coronary ostia, thus decreasing coronary flow. Emboli can obstruct coronary circulation, but due to the protected position of the ostia behind the cusps of the aortic valve, this is uncommon. Emboli may arise from mural thrombi in the left ventricle, left auricle, and left auricular appendage, and from thrombi on arteriosclerotic plaques at the root of the aorta. When coronary embolism occurs as a complication of bacterial endocarditis, the source of the embolus is usually a vegetation on the aortic valve.[1] Most commonly, however, myocardial ischemia is at least partially due to intrinsic coronary artery disease in the form of atherosclerosis.[2] Occlusions generally occur at the site of an atherosclerotic plaque either by superimposed thrombus formation or less commonly by subintimal hemorrhage into a plaque. Rupture of a large atheromatous plaque may allow the pultaceous mass to occlude the lumen completely. Encroachment upon the lumen by a gradually enlarging plaque may eventually result in virtually complete occlusion.

The blood supply to the normal human heart is furnished from the right and left coronary arteries. Normally the right coronary artery extends to the right around the heart in the atrioventricular sulcus and supplies the ventricle and atrium. The left coronary artery divides almost immediately in the anterior sulcus and the descending branch which extends toward the apex in the anterior and the circumflex branch which extends to the left around the heart in the atrioventricular sulcus. In the normal heart there are minute intercommunications between the coronary arteries. In the event of sudden occlusion of a major coronary artery, they cannot prevent infarction. Therefore, the coronary arteries are functionally end arteries although microscopic anastomoses do exist.[3] Thus, if occlusion should suddenly develop in the normal heart, infarction of the area supplied by the occluded vessel would most certainly occur. However, the process of atherosclerotic narrowing is generally a slow one, and thrombosis usually occurs at a site previously narrowed by atheromatous plaques.

Blumgart and his group have demonstrated by their injection and dissection technique that collateral vessels invariably develop around a narrowed coronary segment.[3] Indeed, they noted that interarterial anastomoses are abundant in hearts with long-standing coronary artery disease. One of the causes of the development of these anastomoses is the anoxia that develops in the region that the narrowed artery supplies.

Zoll and his group[4] have shown that anoxia due to anemia or diffuse pulmonary disease results in the

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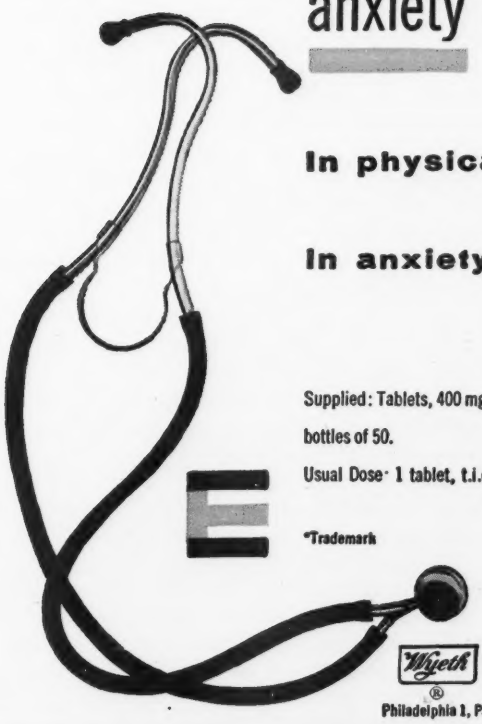
This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Joseph is now serving his internship at Tripler Army Hospital, Honolulu, Hawaii.



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development of coronary collateral in the absence of coronary artery disease. Wiggers feels that the slow establishment of an increased pressure gradient following partial or complete occlusion distends the normally small and useless vessels until they reach significant size.[5] This mechanism is usually quite effective since complete occlusion often occurs in atherosclerotic hearts without giving rise to clinical signs or producing extensive myocardial damage.

Blumgart reviewed 12 autopsy cases which gave histories of uncomplicated angina pectoris. Ten of these revealed old occlusions of two main coronary arteries yet showed no evidence of past myocardial infarction. In another series of 49 cases of uncomplicated angina, an average of 3.5 old occlusions per heart was found with no evidence of infarction.[6] In most of these hearts, focal fibrosis was seen. This would account for the high incidence of death by heart failure in patients with angina. The temporary relative ischemia suffered with each anginal attack or with superadded exercise is apparently sufficient to destroy a few muscle fibers, and thus cumulative loss of muscle eventually leads to enough myocardial weakening to result in failure.

Coronary occlusion and coronary thrombosis are not synomymous with myocardial infaiction. Indeed, the former often is present without the latter, and occasionally infarction may result in the absence of complete occlusion. Only in the heart in which there has been insufficient time for the development of collaterals does single occlusion produce infarction. Nor does the site of occlusion always determine the area infarcted. Thrombosis of the right coronary artery may produce infarction of the left ventricle if narrowing or occlusion of the left coronary artery has produced development of collaterals from the right coronary artery to supply the area previously rendered ischemic. This phenomenon Blumgart terms "infarction at a distance."

Schlesinger[7] has studied variations in the anatomical pattern of the coronary arteries and divided them into three general groups—one with a balanced coronary circulation and the other two with right and left coronary artery preponderance. In the group with the balanced circulation, the right coronary artery supplied the right ventricle and part of the interventricular septum, while the left circumflex and the left anterior descending arteries supplied the left ventricle and the rest of the septum. In hearts with left coronary artery preponderance, the right ventricle was, to an important degree, supplied by the left circumflex and the left anterior descending arteries. In the group showing right coronary preponderance, the right coronary artery supplied a significant portion of the septum and the left ventricle.

The right coronary preponderance group and the balanced group each comprised about 40 per cent of cases, while the group with the left coronary preponderance comprised only 20 per cent of Schlesinger's cases. In the group with left coronary arterial preponderance, the incidence of arterial occlusions was unusually high, and the incidence of infarction was highest. These infarcts generally resulted in death. These findings are not surprising since in such hearts the left coronary artery supplied a relatively large part of the heart and the pattern of the coronary tree was such that development of collateral circulation from the right coronary artery was difficult. Thus the prognosis of a patient following a coronary occlusion or an infarct is significantly influenced by the original vascular pattern of his heart.

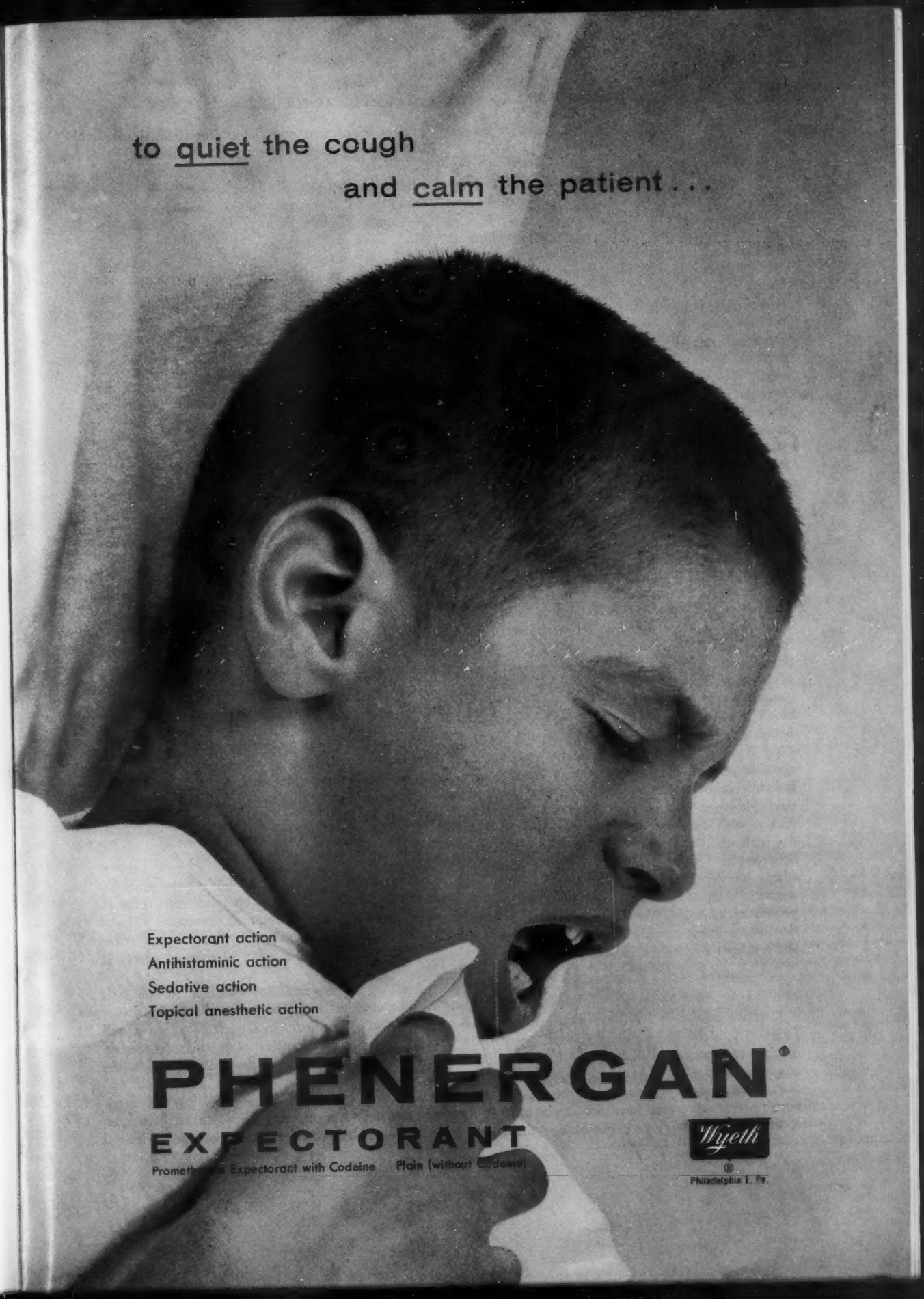
It is apparent, then, that myocardial infarction is usually only one episode in a long process of coronary artery narrowing counterbalanced by the concurrent development of collateral vessels—a race, as it were, between the two processes resulting in varying degrees of ischemia or actual death of cardiac muscle.

Since arteriosclerosis is responsible for coronary narrowing and occlusion, the incidence of myocardial infarction increases with age. It is uncommon prior to the age of 40; and, as with arteriosclerosis, myocardial infarction is much more common in men, occurring about three times as frequently and from five to seven years earlier.[8] Coronary artery disease is also found more frequently in association with those diseases predisposing to atherosclerosis such as diabetes mellitus, primary cholesterol lipoidosis, and hypothyroidism.

In most cases, acute myocardial infarction occurs without relation to effort, but occasionally shock, hemorrhage, unusual exertion, acute hypoglycemia, precordial trauma, and paroxysmal tachycardia may apparently precipitate an attack.

Factors influencing the onset of angina pectoris and myocardial infarction were evaluated by Master and Jaffe.[9] Effort or emotion preceded angina pectoris in almost 100 per cent of cases and acute coronary insufficiency in about 50 per cent. On the other hand, unusual exertion prior to myocardial infarction occurred in only 1.9 per cent. This is explained by the fact that in angina the myocardium has sufficient circulation to maintain it while the patient is at rest, but enough added burden in the form of emotion or exercise may render it ischemic. When infarction occurs, however, it is usually due to thrombus formation, and exercise apparently has little effect on this process. Occasionally prolonged exertion in the presence of ischemia may cause infarction.

A case is cited[8] in which an individual suddenly afflicted with severe chest pain continued to drive his car for 200 miles. The man died one year later of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heart failure, and at autopsy a large healed infarct was seen but no occluded vessel was found.

The relatively large percentage of attacks of coronary insufficiency and infarction that occur during sleep is explaincd by the drop in diastolic pressure at this time.[8] This leads to decreased coronary flow in the narrowed arteries, which encourages thrombus formation. The increased incidence of infarctions occurring in states of low blood pressure or shock is well known.

Acute myocardial infarction may be the first clinical manifestation of coronary arteriosclerosis, but in about half of the patients angina pectoris has been present. The onset of myocardial infarction is classically described as sudden, severe, crushing pain over the precordium, which commonly radiates down the left arm. These patients who have previously had angina immediately recognize the increased severity of the pain, its longer duration, and its failure to respond to rest or nitroglycerine. The pain almost always persists for at least 30 minutes and usually for several hours—a dull ache or heaviness persisting as long as two to three days after the pain.

At the onset of the attack the patient demonstrates the first sequel to the infarct—forward failure. The injured myocardium cannot deliver its usual output, and symptoms and signs of shock appear. The patient becomes pale or cyanotic, is bathed in cold sweat, is dyspneic, nauseated, markedly weak, and apprehensive. Physical examination reveals a cold clammy skin, obvious dyspnea, a weak fast pulse, heart sounds of poor quality, and various arrhythmias, the most common being ventricular premature contractions. The blood pressure begins to fall but, contrary to popular belief, a precipitous fall in blood pressure is unusual. Only 9 per cent of Master's series of patients with acute myocardial infarction showed a rapid drop in blood pressure (to less than 100 mm. Hg. in the first day). However, most of the patients had a fairly rapid decline with the lower levels occurring by the third day, although the lowest levels were reached usually between the 12th and the 20th day.[10] If pulmonary edema has developed, the patient may be markedly dyspneic with audible respiratory wheezes and expectoration of foamy blood-tinged sputum. Auscultation of the lungs reveals widespread medium moist and scattered sibilant "asthmatic" rales.

DIFFERENTIAL DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION

I. *Acute Abdominal Disease Confused with Infarction.* This classical picture is subject to variations. The patient may complain of no pain, or only a dull ache or heavy feeling in his chest, and he may present few physical signs. Ordinarily, however, the diagnosis of acute myocardial infarction can be made easily if the characteristic electrocardiographic changes are present. Confusion with acute abdominal disease is likely when the pain of myocardial infarction is referred to the upper abdomen. Nausea, vomiting, slight distention, fever, muscle spasm and leukocytosis may be common to the two conditions.

Acute cholecystitis, penetrating or perforated peptic ulcer, acute pancreatitis, acute mesenteric thrombosis, renal colic, intestinal obstruction, and acute appendicitis may be confused with acute myocardial infarction. However, the finding of localized pain and tenderness, reflex muscle spasm, and other physical signs of abdominal disorder in the presence of a normal electrocardiogram tends to rule out infarction. Prompt and accurate diagnosis is important lest needless and even fatal abdominal surgery be performed on a patient with myocardial infarction. Conversely, a patient with a ruptured gallbladder or perforated ulcer may be allowed to die if a false diagnosis of acute myocardial infarction is made.

II. *Thoracic Disease Confused with Infarction.* Of the conditions in the thorax which may be confused with myocardial infarction, pulmonary embolism is probably the most common. If a small embolus produces an infarct of the lung, the pain is due to the pleuritis overlying the infarct. However, if an embolus becomes lodged in one of the larger branches of the pulmonary artery, severe chest pain which is indistinguishable from that due to myocardial anoxia may occur, and differentiation must be made from the electrocardiogram. Dissecting aneurysm, hiatal hernia, and mediastinal emphysema may be confused with myocardial infarction or angina pectoris. Therefore, in any case of chest pain, one is duty bound to think of the heart as the source of trouble. A thorough history and physical examination, in addition to electrocardiograms, radiologic examination, and other laboratory tests will usually make possible an accurate diagnosis either incriminating or exonerating the heart.

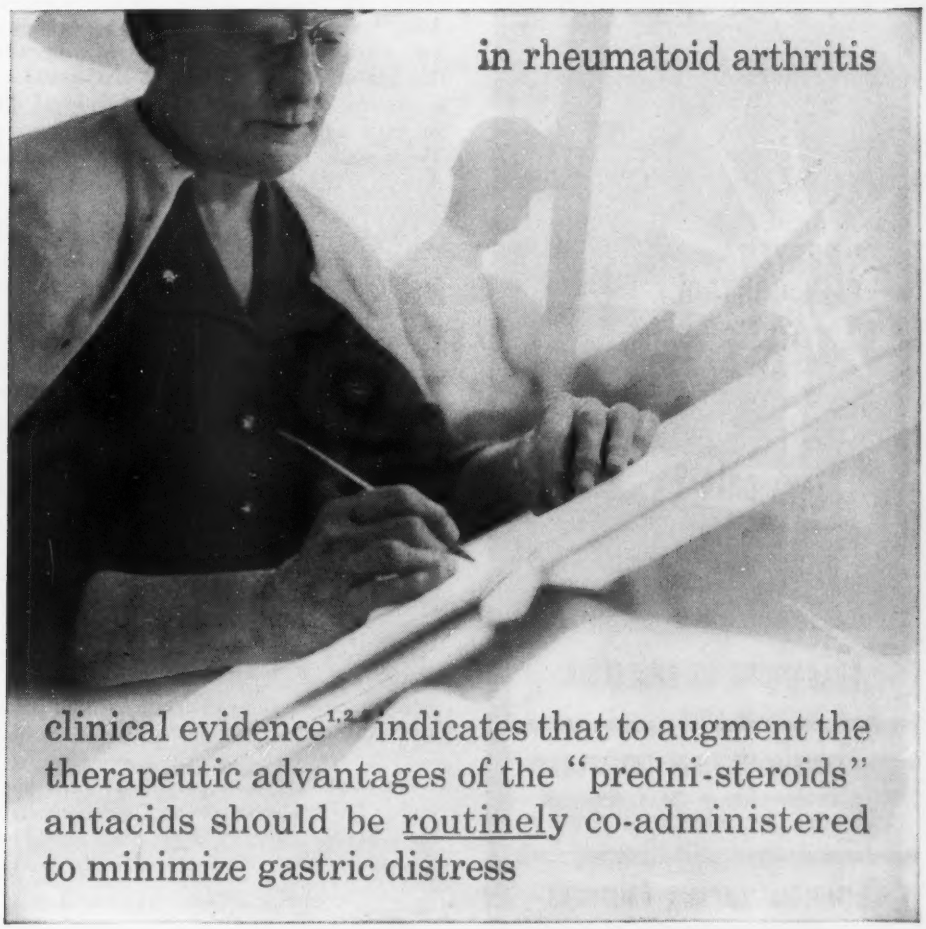
IMMEDIATE TREATMENT OF ACUTE MYOCARDIAL INFARCTION

The immediate management[11, 12] of the acute attack must be directed toward complete rest; relief of pain, apprehension, and dyspnea; prevention of heart failure and deterimental arrhythmias; and active treatment of shock and acute pulmonary edema if present.

I. *Relief of Pain and Apprehension.* Morphine is the drug of choice to relieve pain and apprehension.

II. *Relief of Cyanosis and Dyspnea.* Oxygen is used in all cases where cyanosis or dyspnea is present. Frequently this also lessens the pain and slows the heart rate.

III. *Relief of Congestive Failure.* When early signs of congestive failure supervene (such as moist rales in the lung bases, venous distention, or peripheral



clinical evidence[1,2,3] indicates that to augment the therapeutic advantages of the "predni-steroids" antacids should be routinely co-administered to minimize gastric distress

ROUTINE CO-ADMINISTRATION MEANS

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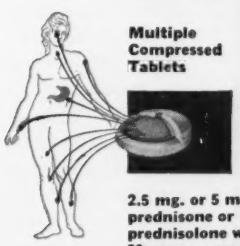

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References: 1. Boland, E. W., *J.A.M.A.* 160:613 (February 25) 1956. 2. Margolis, H. M. *et al.*, *J.A.M.A.* 158:454 (June 11) 1955. 3. Bollet, A. J. *et al.*, *J.A.M.A.* 158:459 (June 11) 1955.

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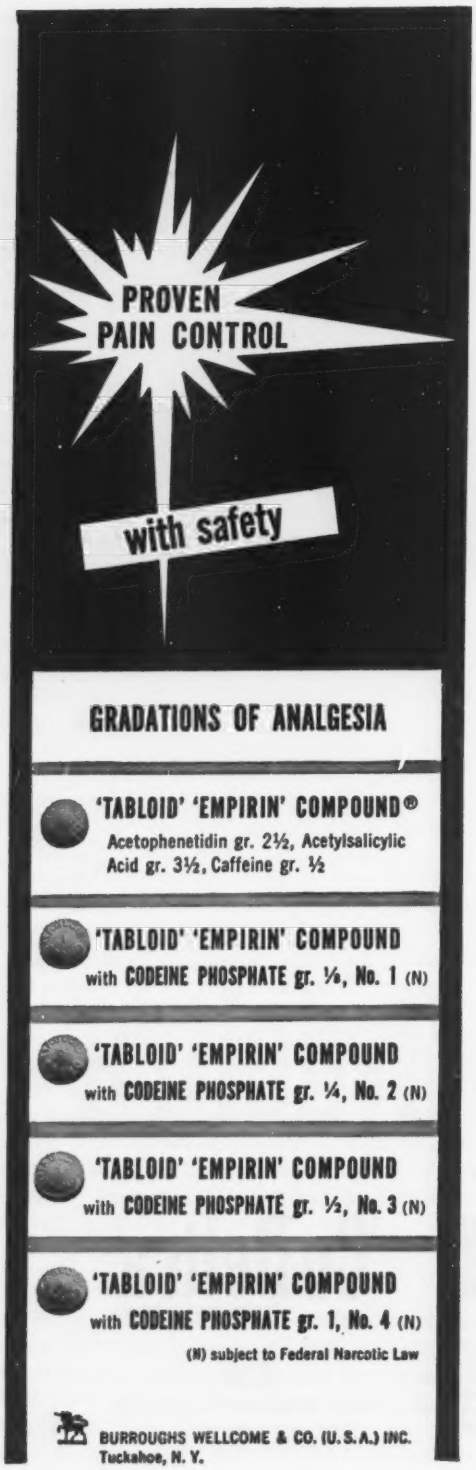
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edema) or with rapid heart rate due to auricular flutter or fibrillation, digitalis should be administered. Askey[13] concluded from his series of 50 patients with myocardial infarcts treated with digitalis that the fear of catastrophic arrhythmias should not be a deterrent to the use of average doses of digitalis for early signs and symptoms of congestive failure. The mortality in his series of 50 patients treated with digitalis was no greater than in his control group of 50 treated without digitalis.

Coronary vasodilator drugs theoretically should be useful to dilate existing collaterals; however, the use of nitroglycerin is hazardous. Administration of this drug may lead to a consequent fall in blood pressure which may precipitate massive infarction and death.

Wegria and his group[14] observed an increase in cardiac output and heart rate without blood pressure change during glycerol trinitrate administration. Cardiac work was therefore increased. These findings suggest that glycerol trinitrate relieves cardiac pain by increasing coronary flow relatively more than the work of the heart. The authors point out that the drug may actually jeopardize the patient with coronary occlusion by increasing the work of the heart without being able to increase the coronary flow proportionately because of the occluded vessel.

IV. *Prevention of Detrimental Arrhythmias.* The routine use of quinidine or procaine amide is advocated by some to forestall development of paroxysmal ventricular tachycardia or fibrillation. However, in some patients quinidine may cause nausea, vomiting, or diarrhea, and both drugs have been known to cause a fall in blood pressure. There is general agreement, however, that one of the drugs should be given if numerous ventricular premature contractions appear.

SHOCK AS A COMPLICATION

Shock is a common and extremely grave complication that occurs during the first 24 hours following infarction. The degree of shock often exceeds, in proportion, the size of the infarct; therefore, it is probable that resolution of the shock state could lead to recovery in a proportion of patients who now succumb. The exact mechanism of cardiogenic shock following myocardial infarction is still obscure. Boyer[14] has reviewed this problem, and he concludes that the mechanisms are different from those operative in traumatic shock. He believes that neurogenic mechanisms may play a role, but that the bulk of the evidence points to inadequate left heart output associated with a redistribution of blood away from the arterial side producing the picture of collapse. With reduced pressure in the aortic arch, coronary blood flow is correspondingly reduced, furthering the production of myocardial ischemia and preventing compensation by the uninfarcted muscle. With further reduction of

cardiac output, more residual blood remains in the ventricle after systole, and backward failure ensues in the form of acute pulmonary edema.

TREATMENT OF CARDIOGENIC SHOCK

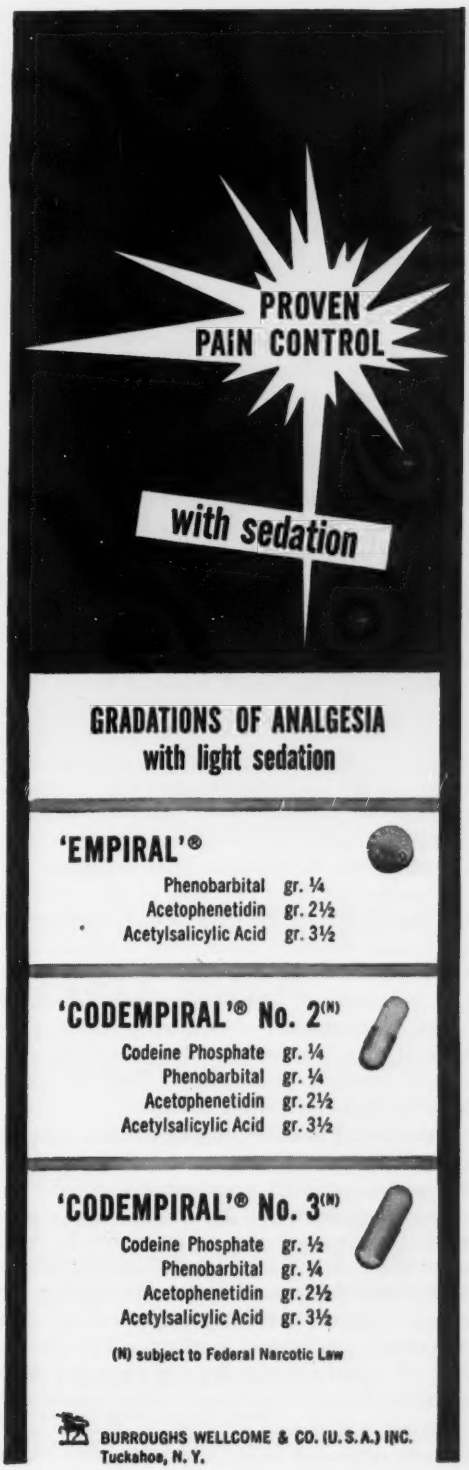
I. *Retrograde Intra-arterial Transfusions Under Pressure.* The treatment of cardiogenic shock is directed toward raising the pressure in the aortic arch to increase coronary flow and thus prevent further ischemia. The aortic pressure is most effectively raised by retrograde intra-arterial transfusions under pressure. This treatment has been found effective in shock so severe that circulation has almost ceased and irreversible damage to such vital organs as heart, liver, brain, and kidneys has been imminent.[15]

Unlike intravenous transfusions, intra-arterial transfusions have been effective with relatively small amounts of blood and have not aggravated pulmonary edema. Indeed, Silber states that the procedure of choice in treating shock where pulmonary edema is present also is the withdrawal of 200 to 400 cc. of venous blood, oxygenating it, and injecting it into the bracheal artery under pressure. Berman[16] and his group also have published good results with intra-arterial transfusions in the treatment of cardiogenic shock. However, lack of readily available special equipment plus the technical difficulties involved have thus far prevented the widespread use of this procedure.

II. *Sympathomimetic Amines.* The use of sympathomimetic amines to effect peripheral vasoconstriction and thus raise aortic arch pressure has proved effective. Miller[17] has reported favorable results with the use of 1-arterenol (Levophed) and suggests that the combination of Levophed and intra-arterial transfusions may be effective in instances where either one alone has failed. Objections have been justifiably raised against the use of epinephrine, which enhances the irritability of the myocardium sufficiently to produce serious ectopic rhythms. Although all sympathomimetic amines in sufficiently large doses will produce ectopic rhythms, 1-arterenol, mephentermine, desoxyephedrine, and others have a greater range of safety than epinephrine.[17]

ACUTE PULMONARY EDEMA AS A COMPLICATION

I. *Mechanism of Acute Pulmonary Edema.* Intravenous blood transfusion, on the whole, has not improved the mortality rate in patients with acute myocardial infarction,[19] and it increases the danger of congestive heart failure. Acute pulmonary edema often develops in the wake of myocardial infarction, and intravenous infusions will encourage this complication by increasing the inflow load to an already weakened heart. According to Harrison and others,[20, 21]



this results in an increase in left ventricular diastolic pressure.

If the weakened heart fails to respond by increased force of contraction according to Starling's Law, secondary back pressure elevation occurs in the left auricle and pulmonary venous system. Ordinarily the pulmonary capillary blood pressure is about 10 mm. Hg. while the oncotic pressure of the plasma proteins is about 25 to 30 mm. Hg. However, the lungs do not have a positive tissue pressure to protect them against outward filtration. On the contrary, the negative intrathoracic pressure of –5 to –10 mm. Hg. exerts a suction on the capillary walls decreasing the margin of safety to around 5 to 10 mm. Hg. If the pressure in the pulmonary capillaries rises enough to overcome this margin, transudation of fluid occurs. This, in turn, produces anoxia, which increases the permeability of the vessels and brings about further edema formation. The resultant asphyxia leads to respiratory stimulation, which in turn lowers the mean intrathoracic pressure, causing further inflow into the right ventricle which, in the presence of a diseased left ventricle, results in more pulmonary congestion and edema. This vicious cycle may lead to death if uninterrupted.

Although this mechanical theory of increased pulmonary capillary pressure has been supported by considerable experimental evidence,[21, 22, 23] certain instances of pulmonary edema following trauma to or disease of the central nervous system, pulmonary infarction, etc., are difficult to explain on this basis. Luisada and others[24, 25] believe that acute pulmonary edema may be explained on a neurogenic basis —either through direct excitation of nervous centers as in skull injury and encephalitis or through reflexes arising from stimuli in the heart or lungs and affecting the permeability of the capillaries.

II. *Treatment of Acute Pulmonary Edema.* Although the mechanism of pulmonary edema is controversial, there is rather general agreement on certain standard methods of treatment. First the patient should be placed in a sitting position.

If the patient in acute pulmonary edema following myocardial infarction has not already been digitalized, ouabain or acetyl strophanthidin should be administered intravenously. This causes an increase in ventricular emptying, with an initial rise in cardiac output and a decline in ventricular diastolic pressure. Consequently pulmonary venous and capillary pressures are reduced, pulmonary congestion and edema are lessened, and dyspnea is relieved. This beneficial effect of digitalis cannot be well explained if the neurogenic theory of pulmonary edema is accepted.

Oxygen in high concentrations is effective in that it lessens respiratory center stimulation, thus decreasing the inflow load to the right ventricle. It also reverses increased capillary permeability caused by anoxia.

Morphine is effective in that it breaks the vicious cycle by dulling the respiratory center and thus alleviating dyspnea, which in turn decreases inflow to the heart.

Peripheral venous tourniquets to reduce circulating blood volume may be used as an emergency procedure, but the danger of peripheral venous thrombosis contraindicates prolonged use of this method.[18] Venesection will accomplish the same purpose.

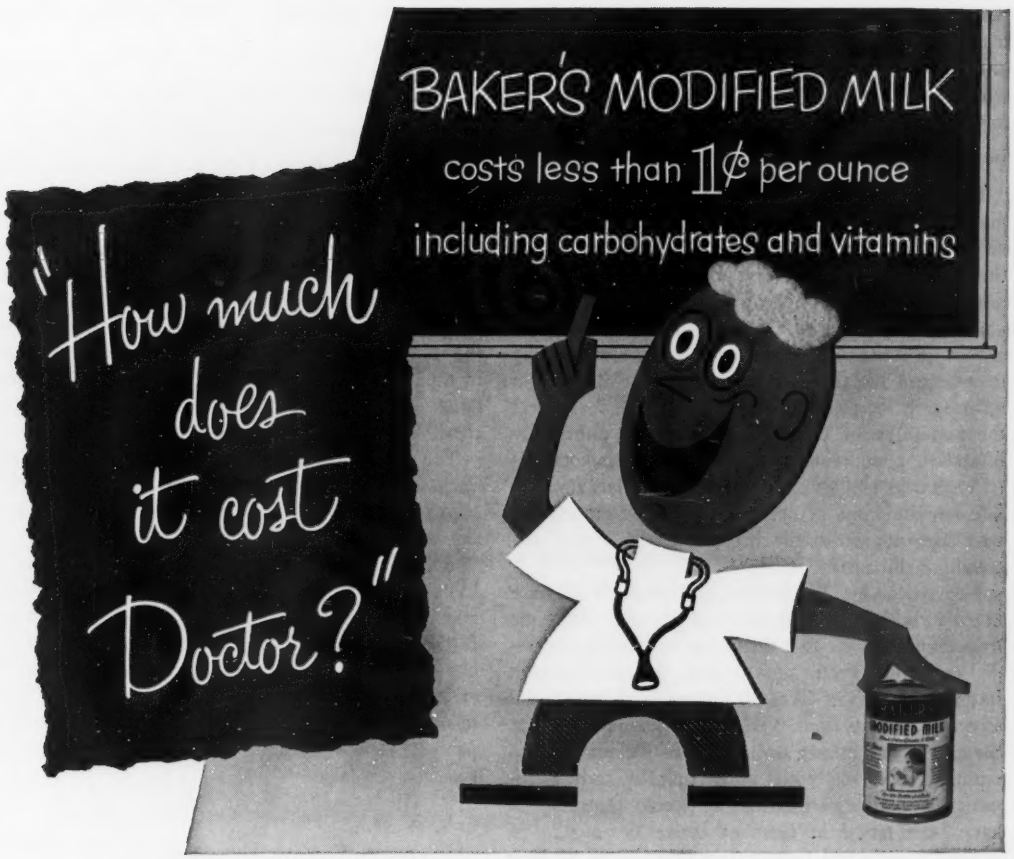
The use of mercurial or other diuretics and a low salt diet reduce edema and thereby tend to prevent nocturnal reabsorption of extracellular fluid. Since such reabsorption is probably the most common mechanism precipitating paroxysmal dyspnea, diuretic drugs are of value in preventing these episodes.[20] Diuretics are indicated whenever the patient exhibits increasing dyspnea or rapid gain in weight.

Positive pressure breathing has recently proved to be of benefit.[26] This tends to diminish venous return to the right heart by abolishing negative intrathoracic pressure and thus reduces the volume of blood in the pulmonary vascular bed. Also it tends to exert an opposing force to outward filtration through the capillary membrane.

EARLY PATHOLOGIC AND ELECTROCARDIOGRAPHIC CHANGES

Although profound changes in hemodynamics may occur and, indeed, the patients may die soon after the infarction, microscopic sections through the infarct show nothing before six hours. Although this is generally true with human autopsies, Karsner and Dwyer[27] noted myocardial changes as early an one-half hour following ligation of a coronary artery in dogs. These changes consisted of poorly outlined areas of congestion, interstitial edema, several small hemorrhages, and slight diminution of transverse striation in the muscle bundles. The reason for this apparent discrepancy is probably that, in human cases, the actual death of muscle cells occurs some time later than the striking clinical picture of chest pain. Existing collaterals may barely maintain the myocardium in an ischemic but living state for as long as several hours.

This also explains why the characteristic QRS changes in the electrocardiogram do not always appear immediately. Electrocardiograms taken during an acute attack show changes corresponding to the histopathologic alterations of the heart muscle. First inversion of the T-wave occurs, characteristic of myocardial ischemia. Shortly following this change, RS-T segment elevation occurs coincident with injury to the myocardium. This current of injury persists sometimes for many weeks, due to extension of the infarct and progressive deterioration of muscle fibers at the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periphery of the infarct. Upon the actual death of the myocardium, the amplitude of the R-wave decreases and a Q-wave appears. Occasionally these latter changes may not appear until a week or ten days following the "clinical infarct."

SIGNS OF TISSUE NECROSIS FOLLOWING INFARCTION

By the end of 24 hours the patient usually has begun to show signs of absorption of products of tissue necrosis. Fever and leukocytosis often appear at this time—the magnitude of each being roughly proportional to the extent of the infarct. The fever is low-grade, usually not exceeding 101° F., and it may not appear until the second or third day. Occasionally the temperature remains normal. Although high fevers occasionally occur, they should lead one to suspect the possibility of complications such as pulmonary infarction, pneumonia, or other infection. Leukocytosis of 12,000 to 15,000 cells generally parallels fever in intensity and time of appearance. The sedimentation rate does not ordinarily become elevated until the second or third day.

Recently Ladue, Wrobeleski, and others[47a, 47b] have noted a rise in the serum levels of glutamic oxalacetic transaminase in the first three days following myocardial infarction. From 2 to 20 times the normal serum levels was found in 49 of 50 patients with transmural myocardial infarction. A rough correlation between the size of the infarct and height of the serum transaminase level was noted both clinically and experimentally. Although increases of serum transaminase have been noted in cases of active destruction of skeletal muscle and liver and in chronic diffuse liver disease, it is unaffected by angina pectoris, coronary insufficiency, heart failure, or digitalis in the absence of active heart cell damage.

PATHOLOGIC CHANGES IN THE HEART AFTER 24 HOURS

I. *Gross Changes.* If death should occur from shock, pulmonary edema, or ventricular fibrillation 24 hours after infarction, the heart at autopsy would show characteristic changes. The infarct would appear as an indistinct area that was paler and drier than normal with small, blotchy, red-purple areas of hemorrhage that are present focally.

II. *Microscopic Changes.* Microscopically the muscle fibers in this area would appear swollen, opaque, and excessively eosinophilic (brick red); and the nuclei could be made out only with difficulty. The cross striations would be diminished and the cytoplasm would have a granular appearance. At this time a slight degree of polymorphonuclear leukocytic infiltration in the interstitial connective tissue at the margin of the infarct would be seen. The margin of the infarct would be irregular since the area supplied by the occluded vessel would interdigitate with another area adequately supplied by collaterals. Infarcted areas might actually be separated by viable muscle if a collateral were supplying an area between two branches of the occluded vessel.

MANAGEMENT OF INFARCTION FOR TWO WEEKS

In the period of time from two days to two weeks following infarction, the clinical picture changes rapidly. Two days following infarction the patient may still be in a state of shock and in critical condition. Friction rub may develop at this time, indicating fibrinous pericarditis. Signs of congestive failure may predominate. However, in an uncomplicated course, the leukocyte count and fever are approaching normal by the third or fourth day, and although the sedimentation rate is still elevated the patient is in much better condition by this time.

The patient's electrocardiogram remains essentially unchanged, except for the previously elevated RS-T segment, which drops progressively nearer the base line during this time.

After the severe pain of acute myocardial infarction has subsided (usually in the first 24 hours), codeine may be substituted for morphine for relief of discomfort. Sedation should be provided with phenobarbital, but deep breathing should be encouraged to prevent atelectasis. A program of massage and passive movements of the arms and legs should be inaugurated to prevent venous thrombosis and periarthritis of the shoulder girdle.

COMPLICATIONS LIKELY TO OCCUR

I. *Cardiac Rupture.* During the period from two days to two weeks after infarction, the amount of necrotic tissue is at a maximum and very little organization has had time to occur. It is apparent that rupture of the heart is most likely to occur at this time, and therefore rest is of particular importance. Should rupture occur, blood fills the pericardial sac and not only causes collapse of the atria and great veins, preventing filling, but also impedes heart action; death occurs almost immediately. White[28] reports an incidence of 3.5 per cent and Gans[29] an incidence of 9 per cent of this complication. In mental institutions, where patients are disturbed, agitated, and unlikely to remain in bed, the incidence is higher.[28]

II. *Thromboembolic Phenomena.* Thromboembolic episodes are among the most dangerous and common complications during this period. Mural thrombus formation may occur as early as 24 hours following infarction, but it is uncommon before the fourth day, after which it becomes increasingly frequent in occurrence up to the end of the first month.[30] The danger of systemic emboli to the brain, kidneys, intestine, spleen, extremities, etc., is obvious. If either the brain or intestine is the site of embolization, death may well result.

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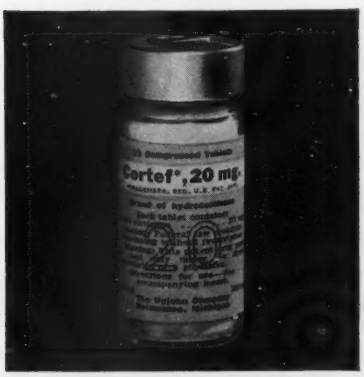

Should an infarct extend through the interventricular septum or involve the right ventricle, the mural thrombus which forms may send emboli to the lungs. More often, the source of a large fatal pulmonary embolus is a thrombus in the veins of the lower extremity or pelvis. Central or peripheral extension of the original coronary thrombus may occur with occlusion of more arterial branches, leading to necrosis of areas contiguous to the original infarct.

ANTICOAGULANT THERAPY

It was in an attempt to prevent these thromboembolic sequelae that anticoagulant therapy was introduced. In 1946 the Committee for the Evaluation of Anticoagulants in Treatment of Coronary Thrombosis with Myocardial Infarction was established. This committee studied intensively 1,031 cases, 442 of which were treated conventionally and 589 of which were also treated with anticoagulants. Following the committee's report of a 33 per cent decrease in mortality with the use of anticoagulants, their routine use was recommended.[31]

Other investigators have emphasized the difficulty of adequately controlling a study of this kind.[34] Doscher and Poindexter[32] pointed out that age, sex, pre-existing hypertension, angina, and previous infarction materially influenced mortality rates and that the state of the heart at the time of infarction must be weighed in any statistical analysis of any therapeutic procedure. Indeed, this therapy is not without danger, and reports of the incidence of major hemorrhages secondary to its use have ranged up to 10 per cent.

Bresnick and his group[33] have concluded from their study of 250 cases that anticoagulant therapy as given in a busy general hospital does not reduce mortality from acute myocardial infarction. They point out many difficulties in maintaining patients at sufficiently stable prothrombin levels, especially by those unskilled in its administration. More recently Rytand[34] has reviewed the various statistical studies and concluded that there is no valid evidence that prognosis for survival is improved by the use of anticoagulants.

Russek and Zohman[35] divided a series of 1,047 consecutive hospital admissions for acute myocardial infarction into "good risk" and "poor risk" groups on the basis of certain prognostic signs. There was an incidence of thromboembolic phenomenon in only .8 per cent of the "good risk" patients. They feel, therefore, that it is unjustified to use anticoagulants routinely since the number of deaths secondary to anticoagulant therapy regularly exceeds .8 per cent. They believe, however, that they should be used in severely ill patients. Since low blood pressures immediately following infarction may lead to extension of the coronary thrombus or formation of a new thrombus, the early employment of heparin in addition to the more slowly acting anticoagulants may limit extension of the original myocardial infarct. Gilchrist[36] used this combination in a well-controlled series and reported a mortality rate reduction from 43 per cent to 21 per cent.

In view of the potential hazards of this therapy, however, one must conclude that only those thoroughly skilled in the administration of these drugs should use them. They should be administered only in a hospital with excellent laboratory facilities, and the recipients of this medication should be thoroughly screened for contraindications. With those limitations in mind, anticoagulant therapy can undoubtedly save lives.

PATHOLOGIC CHANGES FOLLOWING INFARCTION

I. *Gross Changes.* If the patient should die from cardiac rupture, pulmonary or cerebral embolus five days following infarction, post-mortem examination of the heart would reveal further changes. The leukocytic infiltration has progressed, and this can be seen grossly as a yellow-brown, irregular border surrounding irregular pale areas of infarcted muscle. This yellow-brown band becomes broader as leukocytic infiltration becomes more extensive, reaching its maximum width by the sixth or seventh day.

II. *Microscopic Changes.* Microscopically necrosis of muscle is still prominent and is shown by the absence of nuclei and the deep eosin stain of the cytoplasm. At the periphery of the infarct is seen marked polymorphonuclear leukocytic infiltration between and even into the necrotic muscle fibers. This infiltration seems to be limited in extent and toward the center of the infarcted muscle is altogether absent. For this reason, it is of importance to examine the junction of the necrotic with the normal muscle in order to estimate the age of an infarct since centrally the infarct will appear much younger. This leukocytic infiltration is usually more active on the epicardial side of the infarct and in those portions adjacent to uninvolved muscle with good vasculature than at the endocardial edge of the infarct.

By the sixth day many of the polymorphonuclear leukocytes will have become necrotic, and thereafter they gradually diminish in numbers. Newly formed blood capillaries and multiplying fibroblasts can be seen growing into the infarcted area beginning about the fourth day.

There is usually a thin layer of surviving myocardium along the endocardial surface, less than a half millimeter in thickness, which obtains its nourishment from the thebesian veins. Because of the constancy of this finding, Mallory and his group thought it would seem more logical to interpret mural thrombus formation as a result of local dilatation of the heart plus stagnation of the blood in this area rather than as a reaction to the adjacent inflammatory response to the infarct. In many infarcts, particularly those in

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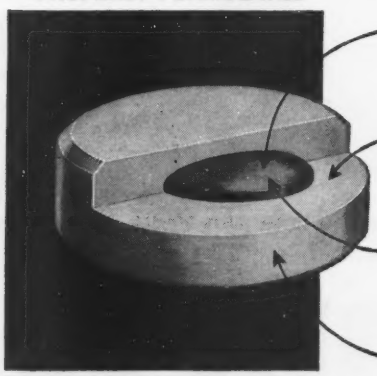

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which the muscle beneath the epicardium is involved, a fibrinous pericarditis can be found after 24 hours. This begins to become organized about the eighth or ninth day.

MANAGEMENT OF INFARCTION AFTER TWO WEEKS

The clinical course from two to four weeks following infarction is in general fraught with fewer dangers than in the acute stage, although thromboembolic complications are commonest during this time. The patient's sedimentation rate is normal, the RS-T segment has returned to the base line, and the T-wave may be upright by this time, although frequently T-wave inversion or flattening remains for years. The Q-wave of muscle death remains. Formerly strict bed rest during this time was advocated; however, the past few years have been marked by a considerable change in thought regarding the value of strict bed rest for the cardiac patient.

Levine and others[37, 37a, 37b, 37c] noted that strict bed rest is liable to aggravate or bring on pulmonary congestion and edema, a variety of gastrointestinal and urinary symptoms, poor appetite, bed sores, and mental depression. There is now evidence that cardiac work is actually greater when an individual in lying in bed than when he is sitting in a chair.[38] While the sitting position permits gravity to mobilize fluid into the dependent parts of the body where it is relatively harmless, the recumbent position encourages the pooling of fluid in the pulmonary circuit where it may be disastrous.[37, 37a]

Levine found that chair rest during the day and elevation of the head of the bed at night would prevent or alleviate pulmonary congestion and edema and also vastly improve the patient's emotional outlook as well as his respiratory and gastrointestinal function. Chair rest was usually begun sometime in the first week of convalescence, and its duration during the day was determined by the patient's ability to tolerate it without fatigue. Although immediate mortality is not increased by such treatment, such a radical departure from the time-honored bed rest will undoubtedly take years to gain general acceptance. Since many patients cannot use a bedpan without undue straining, a bedside commode has been recommended.[39]

PATHOLOGIC CHANGES AFTER TWO WEEKS

I. *Gross Changes.* Should death occur two weeks following infarction, the yellow-brown irregular border characteristic of leukocytic infiltration seen at five or six days will be absent. Instead, granulation tissue is seen as an irregular reddish-purple band surrounding the central mass of unremoved infarcted muscle. The color is due to the presence of newly formed capillaries filled with erythrocytes. At this time the myocardial wall is decreased in thickness due to the actual removal of dead muscle by macrophages. Continued healing results in an increase in the width of the band of granulation tissue, but as it becomes older its collagen content increases, making it appear pale and translucent. At three or four weeks only small islands of necrotic muscle are seen completely surrounded by gray, translucent gelatinous granulation tissue.

II. *Microscopic Changes.* Microscopically invasion into the infarct by young capillaries and proliferating fibroblasts can be seen well at two weeks. By this time, indeed, fine newly formed collagen fibers are present at the periphery of the infarct, and by three weeks the collagen deposition is moderately prominent. The macrophages which have been removing the necrotic muscle are by the 14th day quite numerous and filled with lipofuscin pigment. These pigmented macrophages slowly decrease in number as the infarct becomes older, but they may be found in infarcts more than a year old. Foci of lymphocytes and plasma cells appear with the macrophages but tend to disappear less slowly. A few eosinophils are found at this time.

LATE COMPLICATIONS OF INFARCTION

After four weeks the patient may well resume light activity if his course has been uncomplicated. He is at this time and hereafter faced with the possibility of congestive heart failure, but with restricted activity this may often be avoided. However, the fibrous tissue of the scar is not resilient and contractile, and an aneurysmal dilatation may form as the result of the intermittent force of the systolic intraventricular pressure. This occurs in approximately 10 per cent of all cases of infarction.[30]

Although antemortem diagnosis is infrequent, it is often detectable by fluoroscopy as a bulge with paradoxical pulsation. An RS-T segment elevation persistent several months following infarction has been said to suggest this complication, but Moyer and Miller[40] state that such a pattern may be found with uncomplicated large myocardial scars when there is extensive destruction of the myocardial wall. Such aneurysms, while undoubtedly decreasing the efficiency of the heart somewhat, are apparently of little prognostic import and rarely rupture.[40] Mural thrombi may form in the sac and give rise to embolic phenomena at any time, even years later.

LATE PATHOLOGIC CHANGES

I. *Gross Changes.* The pathologic picture of the myocardial infarct after the fourth week is marked by the deposition of collagen. A small necrotic central mass of muscle may persist in large infarcts after the fourth week. This dead muscle has by this time assumed a pale, red-brown color grossly, and it is seen

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to be surrounded by pale, gelatinous granulation tissue containing considerable collagen. By six weeks collagen is quite a prominent feature in the infarct and becomes more and more dense until the end of the second month when this process has reached its maximum. By the second or third month, the infarct appears as a firm, white fibrous scar, and thereafter it undergoes no further change.

II. *Microscopic Changes.* Microscopically collagen is prominent except adjacent to the necrotic muscle. No acute reaction is seen at this junction, but a few lymphocytes and pigmented macrophages are still present.

SUMMARY OF PATHOLOGIC CHANGES

In summary, then, the following changes have occurred:

I. *First Week.* During the first 24 hours, necrosis of muscle with slight polymorphonuclear leukocytic infiltration at the periphery is seen. From the second to the fourth day, the degree of this infiltration increases progressively. On the fourth or fifth day, the first signs of removal of muscle fibers appear. Blood capillaries and fibroblasts can be found penetrating the infarct from the periphery.

II. *Second Week.* It is during the second week that the removal of dead muscle from the periphery of the infarct becomes a prominent histologic feature. By the tenth day there is a peripheral zone, one mm. or more in thickness, from which the necrotic muscle fibers have been almost completely removed. As a result, numerous pigmented macrophages are found. The ingrowth of blood vessels and connective tissue has become prominent. Moderate numbers of eosinophils, lymphocytes, and plasma cells may be seen. By the end of the second week, the polymorphonuclear leukocytes have practically disappeared.

III. *Third Week.* During the third week, the removal of muscle fibers still continues in large infarcts. Pigmented macrophages are numerous. The first signs of collagen formation may be found in the form of fine collagen fibers at the periphery.

IV. *After the Fourth Week.* After the fourth week, recognition of the age of the infarct is largely dependent upon the amount of collagen that has been formed. The serial changes thus far described were taken from Mallory's classic study.[41] He concludes that small infarcts are almost completely healed after five weeks, whereas large infarcts are completely healed, or undergo no further discernible change, after two months.

PROGNOSIS OF MYOCARDIAL INFARCTION

The immediate mortality rate in myocardial infarction is difficult to determine. Series published in the past two decades have varied from 9 per cent to 51.5 per cent.[9] The average mortality rate is of course important in evaluating different therapeutic procedures, but in an individual case it means nothing. The size of the infarct, cardiac dilatation, previous hypertension, sex, presence of diabetes, shock, cardiac arrhythmias, embolic phenomena, congestive failure, long duration of pain, have all been said to affect prognosis.[42, 43, 44] Therefore, the prognosis and the manner of treatment must be individualized in regard to the history and clinical signs of the individual patient.

However, the prognosis of coronary artery disease and particularly myocardial infarction has been studied in several large series and has been found to be rather less grave than is generally believed. Among 679 autopsies[44] showing old or recent myocardial infarction (of which 20 per cent were not recognized clinically), the average age of death was 67.8 years. Other large series[45, 46] indicate that the majority of patients who survive a myocardial infarction are able to lead fairly normal lives and to be gainfully employed.

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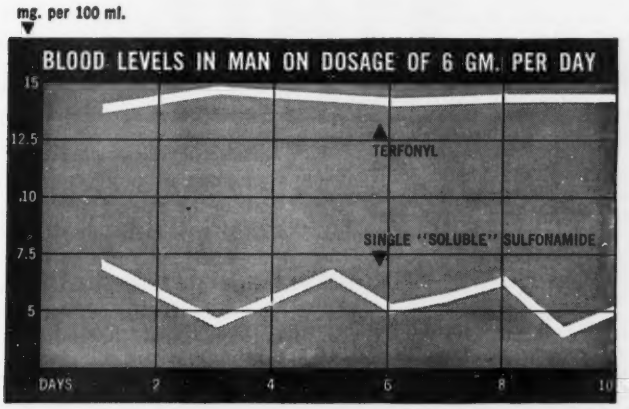

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ANNOUNCEMENTS

The Sister Elizabeth Kenny Foundation announces a program of postdoctoral scholarships to promote work in the field of neuromuscular diseases. Scholars will be appointed annually, and each grant will provide a stipend for a five-year period at the rate of $5,000 to $7,000 per year. Candidates from medical schools in the United States and Canada are eligible. Inquiries should be addressed to Dr. E. J. Huenekens, Sister Elizabeth Kenny Foundation, 2400 Foshay Tower, Minneapolis 2, Minnesota.

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BOOK REVIEWS

Preventive Medicine in World War II. Volume III. Personal Health Measures and Immunization. Edited by Col. John B. Coates, Jr., M.C., and Ebbe Curtis Hoff, M.D. Published by Office of the Surgeon General, Department of the Army, Washington. 394 pages. Price $3.25.

The chapters are on manpower selection, personal hygiene, clothing, nutrition, malnutrition, preventive psychiatry, accidental trauma, and the Army immunization program.

The statistical and research findings that are obtained by the Preventive Medicine Section must be implemented through responsible commanders and staff officers. There is a discussion of some of the Army's problems when a great number of men were needed and standards had to be relaxed.

In the chapter on personal hygiene there is a discussion of body cleanliness, insect repellents, use of safe water and food, and sex hygiene. All of these items relate to the spread of disease and are influenced by the individual soldier's willingness to carry out instructions. A glimpse of the problem that the Army faces in providing bathing and laundry facilities for an army overseas is shown.

When the quartermaster is selecting clothing, the elements of exertion, comfort, and health must be considered. In World War II the problem was increased when armies were sent into hot tropical climates and other armies had to operate in subarctic temperatures. In cold climates one of the most troublesome conditions that was met in the field was "trench foot." Here personal hygiene, shoes, and socks were all factors in preventing this condition.

In the section on nutrition there is considerable discussion of the nutritional value of the "standard ration" and also the nutritional value of the foods that are actually accepted by the soldier. Field rations under combat conditions could not always be assembled in such a way as to assure adequate nutrition for long periods of time.

In the section on preventive psychiatry we find that in World War II, for the first time, an effort was made to distinguish between preventive and clinical psychiatry. Psychiatric screening with classification for jobs was attempted. Group work in mental hygiene was stimulated by pyschiatric personnel in the Army, and some psychiatrists worked with staffs attempting to understand better motivation, morale, leadership, and utilization of men in the Army. It appears that there was no ideal method that provided proper classification and support for the inductee in his early Army training.

Accidental trauma is discussed as historically important in loss of manpower in all previous wars. Non-battle injuries account for many deaths and much disability that occurred in World War II. In the southwest Pacific area during 1943 there were 930 killed in action, 574 non-battle casualties, 152 deaths due to disease, 94 deaths from wounds. About one-half of the deaths in this area were due to non-battle casualties. The accident prevention program in the Army is discussed briefly.

In the chapter on immunization there is detailed discussion about the development and difficulties with yellow fever vaccinations, the adoption of tetanus immunizations, its policy in the use of typhoid, paratyphoid vaccine, the selected use of cholera, plague and typhus vaccinations, and discussion of the Army's use of influenza vaccination.

The book is written by a number of authors, one for each chapter. There are many quotations from reports and bulletins from the War Department and many references not quoted. It is a frank account of some of the problems that the Medical Department of the Army faces in preventing disease and disability. Summaries and conclusions are not common in the book. It is somewhat on the order of "Here are the facts—you can draw your own conclusions." The book probably is best considered as a reference book.—*C.H.M.*

Hunterdon Medical Center: The Story of One Approach to Rural Medical Care. By Ray E. Trussell, M.D. Published by Harvard University Press, Cambridge. 236 pages. Price $3.75.

I would say that this book is a MUST for all of us interested in community hospitals. Certainly this is a most complete record of the trials and errors, triumphs and tribulations in the building and operation of a community hospital. The author formerly served as the director of this hospital in New Jersey, and he is now executive officer of the Columbia Uni-

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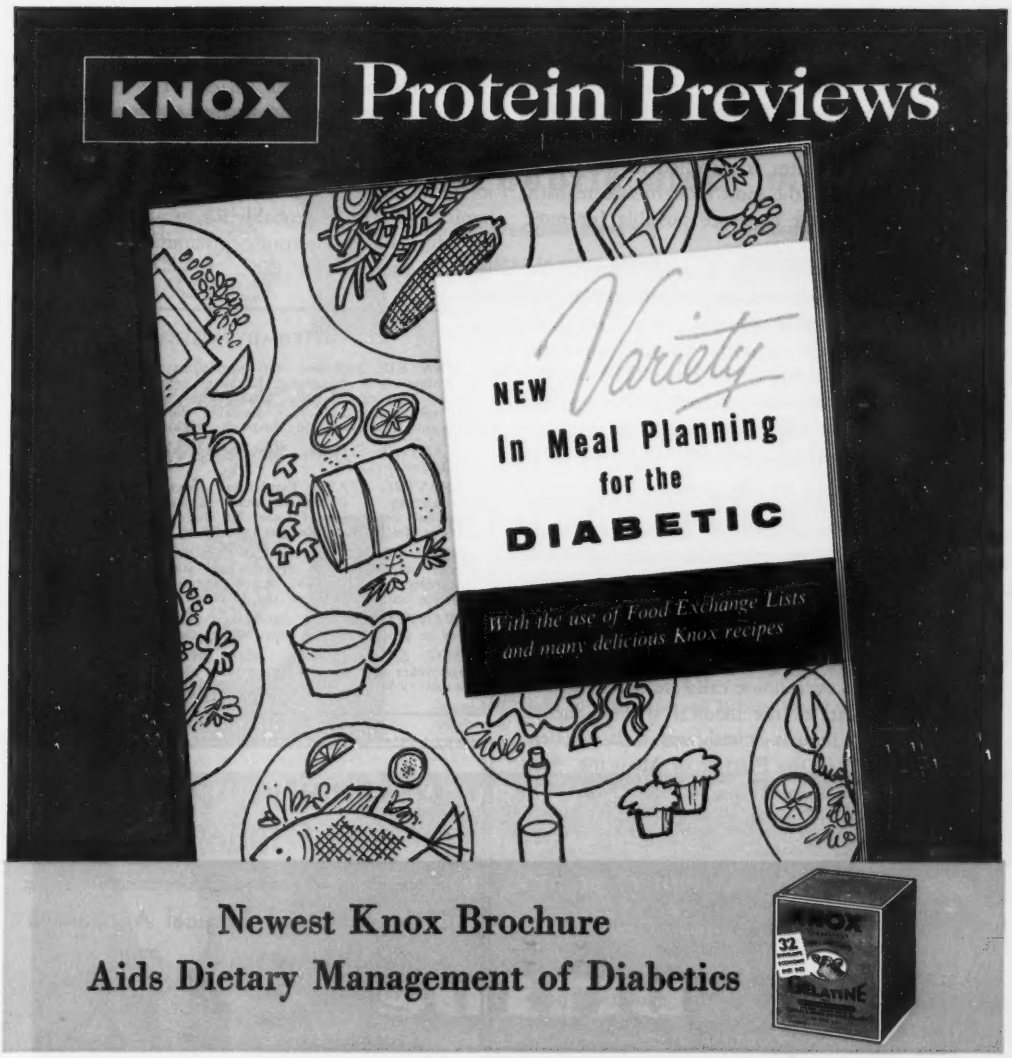

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This is a pattern that could be followed by many rural communities and medical schools, especially since development of the turnpike system of roads and air service. The Hunterdon plan gives the people all the advantages of medical school expert consultation and supervision without sacrificing the personal relationship between the patient and his family physician. By this plan even house calls are not a neglected or forgotten part of the medical system. Such a plan also provides interns or residents with excellent training in the Art of the Practice of Medicine. Such an arrangement as Hunterdon on the part of a medical school should be an excellent internship in general practice.

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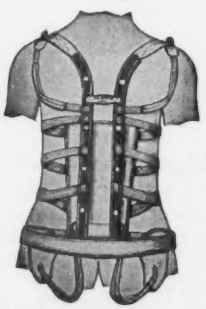

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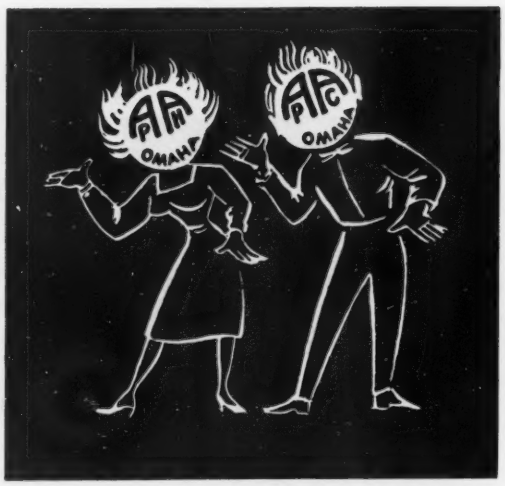

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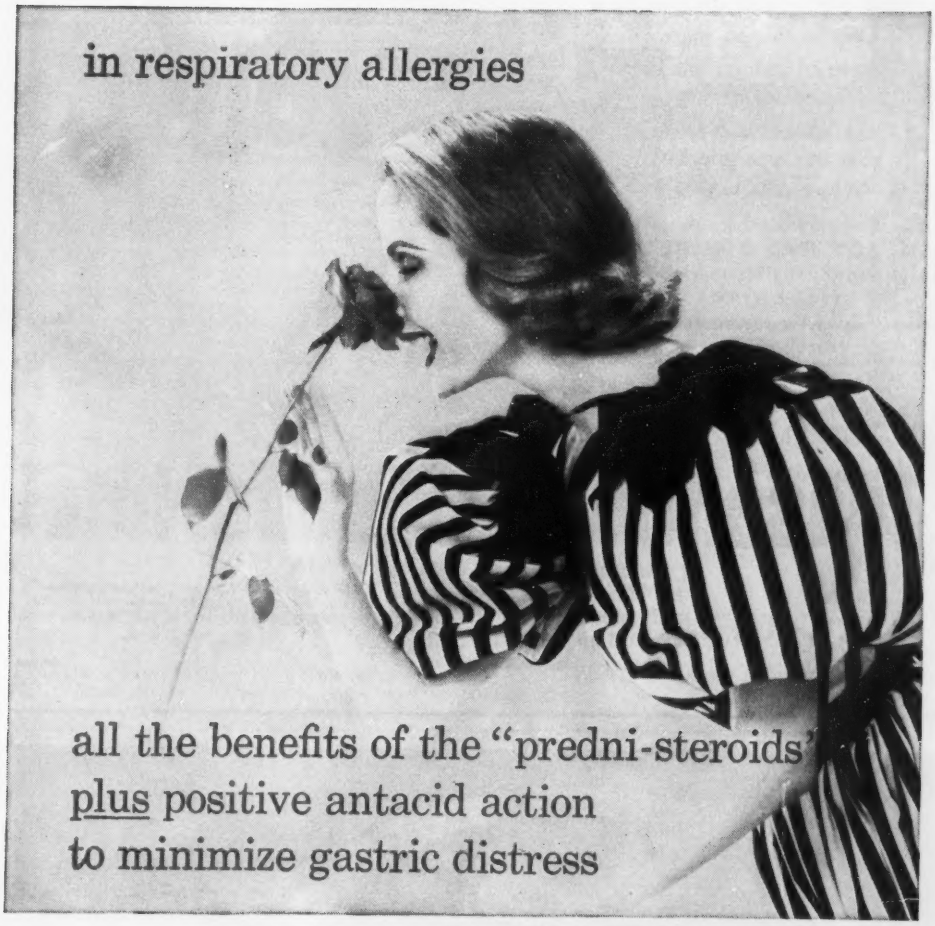

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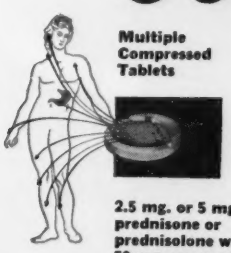

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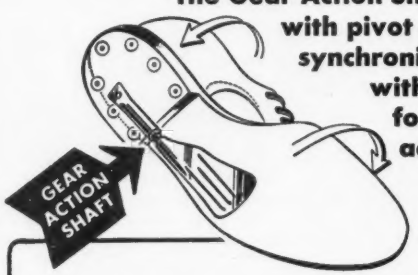

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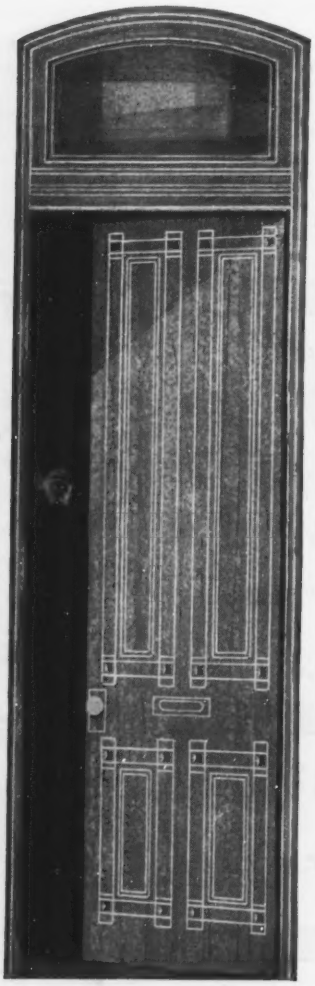

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